STUDIES ON THE PREVENTION OF NEONATAL CALF DIARRHOEA

by

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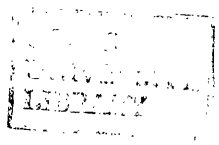

TO MY FAMILY

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DECLARATION

I declare that the work presented in this thesis was, with the exception of the help in various procedures which is fully acknowledged elsewhere, carried out by me.

SUMMARY

Neonatal calf diarrhoea is a major problem in many parts of the world. Most incidents appear to be infectious in origin and a large number of organisms have been incriminated as possible pathogens. However, the majority of outbreaks are probably due to either enterotoxaemic strains of Escherichia coli or rotavirus acting either alone or in combination. The epidemiological features of neonatal calf diarrhoea reflect occasional imbalances between the weight of pathogenic challenge and the level of acquired (colostral) immunity. Treatment usually involves the administration of antibiotics with or without fluid and electrolytes and treated calves commonly die. Prevention techniques usually involve careful attention to colostrum feeding, improved hygiene and the often long-term administration of antibiotics. One possible alternative to this latter approach is to feed various "lactic acid bacteria" in an attempt to establish a balanced intestinal flora and the aim of this series of investigations was to investigate the efficacy of one commercial product, namely LBC concentrate M10 which is a lyophilised formulation of Streptococcus faecium (Cernelle strain 68).

Before each trial commenced the treatment and control groups were carefully balanced in terms of bodyweight and serum immune globulin status; furthermore, group allocations and penning arrangements were such that there was a good chance of an "even" pathogenic challenge. No attempt was made to identify the nature of this challenge although no calf developed diarrhoea due to Salmonella infection. In all 96, 4-7 day old Ayrshire bull calves were used in five separate trials. The results indicated that LBC as a milk supplement for young bucket-fed calves produced definite (and often statistically significant) benefits in terms of less diarrhoea, fewer deaths and greater weight gains.

However, there were strong suggestions that its effects could be overwhelmed with increased pathogenic challenge and were also dependant on the nature of the diet. The results of these studies suggested that the differences in growth rates between test and control calves is due to the effects of diarrhoea in the controls rather than to a growth-promotional effect of LBC in the treatment group.

It was concluded that the feeding of LBC and similar products is worthy of further study since it may well lead to the development of an effective alternative to mass medication with antimicrobial compounds.

CHAPTER 1

NEONATAL CALF DIARRHOEA

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Neonatal diarrhoea (synonyms, calf diarrhoea, calf scour(s), white scour(s)) is a common disease hazard in both dairy and beef herds. By definition (Acres, 1976), it arises some time during the first four weeks of life although the most hazardous period is within seven to ten days of birth (Withers, 1952; 1953). The condition is characterised clinically by varying degrees of diarrhoea and dehydration which often result in depression and weight-loss; when death occurs, it is usually within a few days of the onset of disease and gross post-mortem findings are usually limited to dehydration and a fluid-filled intestinal tract (Radostits, 1965). Neonatal calf diarrhoea is, without doubt, the commonest cause of death during the neonatal period (see Tables 1 and 2, Leech and others, 1968).

HISTORICAL ASPECTS

The problem of neonatal diarrhoea has been recognised in Britain for at least 200 years (Mills, 1776) but the first suggestion that there appeared to be an association between the condition and *Escherichia coli* infection were made by Jensen (1893) following investigations into a particularly severe and persistent neonatal diarrhoea problem on a large dairy farm in Denmark. This, and many similar observations since that time, have lead to the widespread use of yet another term, "colibacillosis" (see below). Neonatal calf diarrhoea was also claimed to be a major cause of death in certain parts of Ireland during the early part of this century (McFadzean and Dollar, 1903) and somewhat later, a study of certain selected (i.e. problem) herds in Scotland revealed that neonatal mortality, that is

Sign of ill health	Number of observations	Per cent of total observations	Per cent of calves affected
Scouring	2,800	47.0	8.9
Coughing	1,202	20.1	3.8
Off-feed	571	9.6	1.8
Fever	232	3.9	0.7
Inco-ordination	104	1.7	0.3
Other signs	1,058	17.7	3.4
TOTAL	5,967	100.0	-

Table 1. Number of observations of ill health recorded and percentages of calves affected; concurrent signs of ill health in the same animal are counted independently. (Leech and others, 1968)

Types of disorder	Percentage of the carcasses examined
Septicaemia	24.8
Gastro-intestinal	44.9
Respiratory	10.8
Others	15.6
Not identified	3.9
TOTAL	100.0

Table 2. Types of disorder found at post mortem examination of 350 survey calves. (Leech and others, 1968)

mainly following diarrhoea, ranged from 20.6 per cent to 23.7 per cent (Jordan, 1933). Later surveys, which were carried out on larger populations of cattle and not restricted to "problem farms", revealed that calf mortality up to six months of age in England and Wales was 5.5 per cent (Lovell and Hill, 1940) and 6.8 per cent (Withers, 1952; 1953) and in Scotland, 11.4 per cent (Lovell and Hill, 1940) and 11.9 per cent (Withers, 1952; 1953); 48 per cent of deaths in the studies by Withers (1952; 1953) occurred within the first week of life and in his view, the commonest cause of death was "colibacillosis". This view would be supported by the findings of a study carried out by Gibson (1961) on dead calves submitted for post-mortem examination to Veterinary Investigation Centres in England and Wales, 1959-1960 (Table 3) although, clearly, some doubt must now exist as to the accuracy of diagnosis vis-a-vis "colibacillosis".

Many factors have been shown to affect the rates and patterns of neonatal calf mortality. Withers (1952, 1953) was first to demonstrate the important effect of how colostrum is first administered; when this is from a pail the mortality rate is three times higher than if newborn calves first obtain colostrum by natural suckling. Other factors (Table 4), such as season (Leech and others, 1968; Oxender and others, 1973) and place of birth (Leech and others, 1968; Selman and others, 1970) would seem merely to reflect a calf's chances of first obtaining colostrum by natural suckling (Selman, 1973). There is also a breed factor involved in neonatal calf mortality (Withers, 1952; 1953) although on many occasions this may simply reflect different forms of husbandry (Selman and others, 1970).

In a survey carried out about ten years ago, it was estimated that each year in Britain, 89,000 live-born, colostrum-fed calves die

Age	Number of calves examined	E. coli infection	Salmonellosis	Other findings
Under 3 days	107	54 (50%)	3 (3%)	50
3-7 days	255	144 (56%)	10 (4%)	101
1-4 weeks	807	213 (26%)	157 (19%)	437
1-3 months	551	74 (13%)	126 (23%)	351
3-6 months	273	21 (8%)	12 (4%)	240
TOTAL	1993	506 (25%)	308 (15%)	1179 (59%)

Table 3. Age incidence of E. coli infection and salmonellosis in calves (Gibson, 1961)

Place of birth	Winter (Nov.62 to Apr.63)				Summer Oct.62 and May to Sept.63	
	Jan. - Feb.		Other months			
	per cent deaths	No. of deaths	per cent deaths	No. of deaths	per cent deaths	No. of deaths
Calving box or cowshed	4.03	158	4.28	294	1.96	78
Field	4.90	22	2.76	62	1.18	87
Yard or other place	7.49	47	4.21	47	1.80	8

Table 4. Percentage mortality in the first month of age and numbers of deaths, showing effects of season and place of birth. (Leech and others, 1968)

(Leech and others, 1968); the economic loss has not been estimated but must stand at many millions of pounds. In Scotland, it has been estimated that around 14,000 dairy heifer calves died annually (Selman, 1969) but again the cost has not been assessed.

DISTRIBUTION

The geographical distribution of neonatal calf diarrhoea and mortality has been discussed by Lovell (1955) and Sojka (1971) who both have emphasised that the condition has been recognised and documented in many different countries including Africa, Asia, the Americas, Australasia and Europe. In general, the situation in more temperate areas are similar to those described for Britain. Usually neonatal calf diarrhoea emerges as a significant problem in developing countries when livestock intensification takes place although it should be noted that little information is available regarding neonatal losses under less intensive conditions in these countries.

Since a number of detailed studies have been made in the United States, certain of the findings should be emphasised. Estimates of neonatal mortality rates range from eight to 25 per cent (Lassiter and Seath, 1955; Grunsel, 1956) and the financial loss (1964) has been estimated for both beef and dairy cattle as well in excess of $ 40M (Amstutz, 1965) but when 1973 values are considered, losses might well exceed $ 200M (Oxender and others, 1973). One study on heifer calves in New York State (Hartman and others, 1974) revealed that in dairy (Holstein) herds, mortality was not affected by diet (i.e. whether calves were fed milk or milk substitute) but did increase with increasing herd size. Another finding which is of particular interest in view of the "personal factors" mentioned by Withers (1952, 1953) was that the mortality rate apparently depended to a great extent

on which particular member of the unit was caring for the young calves (Table 5); calves tended by mothers or wives had almost twice the survival chances than calves tended by hired labour (Oxender and others, 1973).

CLINICAL AND EPIDEMIOLOGICAL FEATURES

Modern epidemiology is based upon the premise that diseases in populations have multiple determinants and that these usually include one or more specific infectious agents as well as factors associated with the hosts and their environments (Schwabe and others, 1974) and certainly such a situation has long been suspected as occurring in neonatal calf diarrhoea.

Salmonellosis apart, neonatal calf diarrhoea may assume several different clinical forms (Gay, 1965). In one form, a syndrome generally associated with colisepticaemia, progression to death is usually so rapid that diarrhoea is minimal or absent (Gay, 1965). In the enteric toxaemic form of the disease there is usually profuse diarrhoea and rapid prostration and death not infrequently supervene. The syndrome arises as the result of proliferation of certain specific strains of *E. coli* within the gut lumen; death is due to the absorption of enterotoxins (Gay, 1965). In retrospect, it is now clear that the syndrome which was described by Gay (1965) as "enteric colibacillosis" is very probably of mixed or alternative aetiology. Certainly, the early experimental work which incriminated *E. coli* with this last syndrome (i.e. in the absence of specific enterotoxin-producing serotypes of *E. coli*) only described quantitative changes in the counts of these organisms in the upper small intestine (Carpenter and Woods, 1924; Smith and Orcutt, 1925). Thus, while in certain cases, *E. coli* infection may be the prime reason for the problem, it is very probable

Person(s) caring for calves	No. of farms	Mean herd size	Calf mortality (per cent)		
			Winter	Summer	Annual
Operator	171	46.0	16.2	10.0	12.8
Hired labour	25	53.2	28.1	12.4	20.1
Mother or wife	25	38.2	15.0	9.4	12.3
Children of operator	66	44.1	16.8	10.0	13.1
Operator with assistance	67	46.1	16.2	10.9	13.5
All other combinations	24	44.9	16.3	11.1	13.4

Table 5. Relationship between person(s) caring for calves and calf mortality on 378 dairy farms (Oxender and others, 1973).

that in others, rotavirus (Woode and others, 1974) or some other viral agent is of major importance. Indeed it was once suggested, on the basis of inconsistent bacterial isolations that the syndrome called enteric colibacillosis by Gay (1965) might well be caused by "nutritional factors" (Smith, 1962). Nevertheless, even in this latter syndrome, gross metabolic derangements may arise due to severe diarrhoea and this may lead to a metabolic acidosis-induced primary acute heart failure and death (Fisher, 1965).

Early descriptions of neonatal calf diarrhoea did not, of course, resort to the type of subdivision suggested later by Gay (1965). However, if only for their clinical detail, it is worthwhile paying them limited attention.

"Acute cases of white scour frequently terminate in septicaemia and in such cases the calf may be found prostrated. Death occurs within a few hours. In the less acute form of the disease the calf becomes very dull, refuses to suck and may stand with head down and a fixed expression. Diarrhoea then usually follows, the faeces have a penetrating and unpleasant odour, are greyish-white, and sometimes contain gas. Consistency of faeces is usually fairly firm at first but becomes steadily weaker, the coat becomes rough and the eyes sunken, the animal has a depressed appearance and tends to lie a great deal in a stretched-out position, being reluctant to rise. If forced to its feet the calf stands with its back arched, exhibiting abdominal pain, straining and passing liquid faeces which sometimes may contain blood-stained mucous" (Smith, 1934).

"In some cases the diarrhoea continues and in others the scouring is intermittent. The affected animal becomes very weak, often showing evidence of dehydration, temperature becomes subnormal

and the calf finally becomes prostrate with cold extremities and mouth. Death usually follows rapidly at this stage after the state of coma. Recovered calves frequently show a pot-bellied appearance and there is often loss of hair round the muzzle, behind the ears and down the legs" (Disease of farm livestock, 1957).

"Some cases show pneumonic symptoms and sometimes, in protracted cases, tenderness and slight swelling of the joints are noticed" (Jordan, 1933).

A later observation might also be included here; "Meningitis was also common in calves with or without intra-ocular abscesses" (Mosher and others, 1968).

There is little doubt that the classification suggested by Gay (1965) greatly simplified the clinical situation in what he termed "colibacillosis". However, the probability that the aetiology of his "enteric colibacillosis" form of the disease is not simply E. coli infection should be borne in mind. A summary of syndromes reported in a questionnaire survey by Leech and others (1968) emphasises the high prevalence of diarrhoea (Tables 1 and 2).

While a full discussion of the mechanism involved in the absorption of colostral globulins by newborn calves and the importance of this phenomenon to their health and well-being is dealt with in the next chapter, the subject deserves brief consideration here because of the marked effect variations in colostral globulin absorption exert on the epidemiology of neonatal calf mortality and diarrhoea.

Briefly, calves are born agammaglobulinaemic and are dependant upon acquiring antibodies from their dam's colostrum. The importance of colostrum in conferring resistence to certain neonatal

disease was demonstrated almost 60 years ago (Smith and Little, 1922) although it has already been emphasised that vast numbers of colostrum-fed calves die annually in Britain (Leech and others, 1968). The indications are that the bulk of these calves, although colostrum-fed, have failed to absorb immunologically adequate quantities of antibody; in other words they are agamma or hypogammaglobulinaemic despite their having been fed colostrum (Selman, 1969; 1973). Many surveys have now shown that a marked individual variation exists in the ability with which calves absorb colostral globulins (Gay and others, 1965; McEwan and others, 1970; Selman and others, 1970) and the factors which have been shown to significantly affect absorption efficiency under practical/farming conditions have been defined by Selman (1969; 1971; 1973). These are the timing of the first feed of colostrum, the mass of globulin presented, the presence of the dam and the breed of calf (although under practical farming conditions it would seem that this latter factor is of considerably less significance than the first three). Furthermore, it has been suggested (Selman, 1969; 1971; 1973) that the varying patterns of neonatal mortality and diarrhoea (especially those due to season, place of birth and method by which colostrum is first presented) merely repeat varying uptakes in colostral immunoglobulin as a result of varying abilities of calves born under differing conditions to first obtain colostrum by natural suckling.

POST-MORTEM FINDINGS

In calves which have died from the septicaemic form of colibacillosis, there may be no sign of diarrhoea having occurred although quite often scanty semi-solid faeces may be found staining the tail and perineum (Gay, 1965). Other signs may include splenic

enlargement, Petechial haemorrhages on spleen, heart, kidney and thymus and excessive fluid in joint-cavities, sometimes containing clots of fibrin (McEwan, 1968).

In calves which have died as the result of severe diarrhoea, the gross post-mortem findings are often limited to signs of marked weight-loss and/or dehydration and faecal staining of the tail and hindquarters. In newly-dead animals there is usually no marked discoloration of the intestine and the other viscera appear normal; there is often distension of the urinary bladder. In cases which have survived for several days there may be signs of an acute exudative pneumonia which is usually assumed to have arisen as the result of hypostatic congestion (Obi, 1980).

Since histopathological studies are hardly ever carried out in such cases and also the detailed information which exists is mostly based on examination of experimental cases, this aspect will be discussed later.

AETIOLOGY

For many years, calf diarrhoea has been considered to be a somewhat complicated condition. While it has been emphasised that diarrhoea *per se* should be thought of as merely a clinical manifestation of several different diseases (Lovell, 1955), most authorities have tended to regard neonatal calf diarrhoea as primarily an infectious disorder which exerts its most severe effects in agamma or hypogammaglobulinaemic calves, particularly in the face of adverse environmental, managemental or nutritional states. However, over the last 10-15 years there has been a distinct change in emphasis regarding the nature of the infectious agent(s).

Early research work into enteric disease was largely directed towards studying the bacterial populations of calf intestine (Jensen, 1893; Lovell and Hughes, 1935) and as a result it became accepted that by far the most significant cause of neonatal calf diarrhoea was the bacterium, Escherichia coli. However, in more recent times, largely as the result of investigations originally carried out in the United States (Mebus, 1975) an association between neonatal calf diarrhoea and certain virus infections have been highlighted and while it is now widely held that either class of organism may be responsible for outbreaks of neonatal calf diarrhoea, neither the relative prevalence of the two infections nor the possibility that they may act in concert to the detriment of a young calf appears to have been investigated widely in the field.

The following section reviews the literature regarding (i) bacteria, (ii) viruses and (iii) predisposing factors associated with neonatal calf diarrhoea. However, most emphasis at present regarding aetiological agents is on E. coli and rotavirus infections since current information would suggest that these are the most significant micro-organisms in this syndrome and these two infections are dealt with in most detail.

(i) Bacteria

Although Jensen (1893) was prepared to suggest that the bacteria which he found in the faeces of diarrhoeic calves were actually causing that problem, later findings that such organisms were also present in the stools of healthy calves made it quite clear that the condition was not a simple infectious process. Eventually it was established that many different strains of E. coli existed and much effort was directed towards typing these strains, particularly using

serological (sero) typing techniques presumably in the hope that clear distinctions could be made between the E. coli serotypes of diseased and healthy calves.

The identification and classification of these various serotypes is dependant upon the recognition of heat-resisting "O" (somatic) antigens, "K" (Capsular) antigens and, to a lesser extent "H" (Flagellar) antigens in those strains which are motile. The "K" antigens may be subdivided further into "A", "B" or "L" forms according to their relative heat-stability. The diagnostic scheme of Kauffmann-Knipschildt-Vahlne (1947) included 126 "O" groups which were subdivided according to their "K" and "H" antigens. This scheme has gradually been enlarged (Edwards and Ewing, 1962) and fairly recently, in a review of the situation, Sojka (1971) claimed a total of 146 "O" antigens which sometimes exhibited cross-reactivity (i.e. antigenic relationship) between themselves and also other antigens of other members of the class Enterobacteriacae. The same author stated that to that date a total of 91 "K" antigens and 49 "H" antigens had been identified.

Using such a scheme, many attempts have been made to identify "pathogenic" types of E. coli and such an approach has met with a certain degree of success at least in some of the forms of colibacillosis (Gay, 1965). Certain workers (Smith and Orcutt, 1925; Lovell, 1955) felt that in young calves, a delicate balance existed between the various serotypes of E. coli and other organisms which were present on the mucous membrane of the alimentary tract and when this was upset in favour of E. coli, diarrhoea occurred. The same authors felt that whether or not death occurred depended upon the strain(s) of E. coli which were present and "other (host) factors"; they recognised that lack of colostrum was an important contributory factor but suggested

that immunological specificity of colostrum was equally important.

Healthy and diarrhoeic calves may harbour similar E. coli populations in their intestines (Smith, 1962). However, it has been shown that certain strains of E. coli may increase in virulence after producing septicaemia or diarrhoea (Amstutz, 1965) and this has prompted other workers (Glantz and others, 1972) to suggest that a "healthy" balance may be upset so that eventually a "pathogenic" strain may become dominant and thus give rise to an outbreak of neonatal diarrhoea. That this situation may be responsible for the infection "build-up" in a calf house has also been suggested by Wood (1955).

Certain other techniques have been devised to attempt to assess the pathogenicity of different E. coli serotypes in vitro. Perhaps the most interesting are the studies of Smith and Halls (1967) which investigated the ability of different serotypes to dilate ligated intestinal loops following the release of enterotoxin(s). This work was later developed (Bywater, 1970) and dilatation was quantified in terms of the dynamics of fluid transfer across the intestinal epithelium. From these studies it would appear that only certain serotypes are capable of producing pharmacologically-significant quantities of enterotoxin(s) (see below).

In one attempt to clarify the situation regarding the role of E. coli in neonatal calf diarrhoea and disease (Gay, 1965) it was suggested that, as a first step, care should be taken to define the form of colibacillosis being encountered or investigated. The author, himself, defined three forms of calf coli-bacillosis (i) colisepticaemia, in which systemic invasion of certain strains of E. coli arises, causing bacteraemia and usually death within 48 hours

(ii) enterotoxaemic (or iso) colibacillosis, in which infection by certain "mucoid" strains of E. coli is limited to the intestinal tract but where the release and absorption of "exotoxins" (sic) produces severe diarrhoea, collapse and often fairly rapid death and (iii) enteric colibacillosis in which localised (alimentary) infection occurred with a multiplicity of strains of E. coli, a variable degree of diarrhoea occurred leading to death in only the most severe cases. Each of these forms will be discussed in turn:

Colisepticaemia: In this form of colibacillosis, diarrhoea may or may not occur depending on the survival time. A high but transient fever may be detected, soon to be followed by prostration and, very commonly, death. At necropsy, petechae may be found in many organs but especially the kidneys, spleen and lungs; the joints may be swollen with increased amounts of synovial fluid. Meningitis is also common (Fey, 1972). The disease is limited to the first week of life under both field and experimental conditions and is the usual cause of death in agamma or markedly hypogammaglobulinaemic calves.

The bacterial findings in this syndrome have been reviewed by Gay (1965) and Fey (1972) who both commented upon the extraordinary variability of E. coli serotypes involved. This, and the finding that the common colisepticaemia strains are also those which are most frequently isolated from healthy calves (Kaeckenbeeck and Thomas, 1960) strongly suggests that almost any strain may be capable of systemic invasion given the right opportunity (i.e. an agamma or markedly hypogammaglobulinaemic calf).

Enterotoxaemic Colibacillosis: This form of colibacillosis is characterised by a sudden onset, and severe diarrhoea usually, but not always, in calves under one week of age. Sudden, profound changes in

serum electrolyte levels occur; marked haemoconcentration ensues and death is a common sequel. At necropsy, dilated intestinal loops are usually the only significant findings and E. coli serotypes are to be isolated only from the small intestine and associated lymph nodes (Logan and Penhale, 1972).

Certain strains of E. coli are regularly associated with this syndrome, namely those strains which possess an "A"-type "K" antigen (Gay, 1965). The ability of the enterotoxin(s) produced by those serotypes to dilate intestinal gut loops (Smith and Halls, 1967; Bywater, 1970) has focussed much interest on the local effects of these compounds.

Enteric Colibacillosis: This form of colibacillosis in calves was defined by Gay (1965) as a diarrhoea syndrome of variable severity and mortality due to localised (i.e. intestinal) infection by E. coli serotypes normally present in a calf's environment. In the past there has been a tendency to include in this category, all individual problems or outbreaks which have not conformed to the two forms described above (i.e. colisepticaemia and enterotoxaemic colibacillosis) and this, together with the multiplicity of serotypes which are deemed to be capable of producing this latter syndrome and the confusing results of many bacteriological investigations have made this by far the most controversial forms of calf colibacillosis. Unfortunately, limited studies (McEwan, 1968) would suggest that it is by far the commonest syndrome even if it is not a single disease entity.

No doubt because of the difficulties involved in setting experimental models of "enteric colibacillosis", this particular syndrome has not received the degree of attention which the other

forms have received despite its prevalence; consequently the strain situation is largely undefined (Gay, 1965). Early studies (Smith and Orcutt, 1925) revealed that E. coli counts were much higher in the anterior small intestine and abomasum of diarrhoeic calves than in healthy animals. However subsequent investigations (Smith, 1962; Smith and Halls, 1967) have cast doubt upon the real significance of these claims since high "anterior gut" counts may be present in some healthy calves. Moreover, Smith (1971) was prepared to suggest that the findings of most early workers were mainly due to post mortem changes. The inconsistent bacterial findings in the enteric colibacillosis syndrome also prompted Smith (1962) to state that there was no evidence to support the view that E. coli was involved in any syndrome other than colisepticaemia and enterotoxaemic colibacillosis; he went on to suggest that the likely cause of the "enteric colibacillosis syndrome" was nutritional rather than microbiological. The same or similar consideration have given rise to many varied suggestions regarding the importance of predisposing factors (see below) and also to claims that E. coli infection may only become established following damage to the intestinal wall by viruses (Stair and others, 1972). Nevertheless, it has to be recognised that this syndrome would appear to be the commonest form of neonatal calf diarrhoea, at least in the West of Scotland and epidemiologically (Selman, 1973) and immunologically (McEwan and others, 1970) behaves like an infectious disease. It should also be recognised that this whole classification was evolved largely before the possible significance of viruses and especially rotaviruses was realised; thus, many of the equivocal results regarding the bacterial findings in "enteric colibacillosis" might well be explained on the basis that the syndrome is at least in part due to a virus infection. It is

perhaps of more than academic interest to ask oneself on what criteria were the diagnoses of neonatal colibacillosis made by earlier workers such as those outlined in Table 3.

Although E. coli is the organism which is generally associated with neonatal calf diarrhoea, other bacteria have sometimes been suspected or shown to have been involved in the problem. In England and Wales, Sojka and Field (1970) found that 64.3 per cent of all salmonellosis diagnoses made by Veterinary Investigation Centres involved calves although the precise age incidence was not given. However, in another study, (Rothenbacher, 1965) it was shown that the average age of 51 calves which died from acute or subacute salmonellosis was 13.7 days; most of these had developed diarrhoea between three and five days of age (range 2-42 days).

Neonatal salmonellosis has been described in two forms, a septicaemia and an enteritis. The clinical features of the former syndrome are similar if not identical to those of colisepticaemia (Rokey and Ehrling, 1959; Smith and Rutherford, 1965). The enteritic form is characterised by diarrhoea and fever, commonly with blood mixed with the diarrhoeic stools. The condition is often fatal at this age (Hibbs and Foltz, 1964; de Jong and Ekdahl, 1965). In calves which do survive the earlier stages of the disease, there is often a persistent diarrhoea but if calves die of salmonellosis, the majority do so within two weeks (Rotherbacher, 1965; Robinson, 1966). In both instances, diagnosis of salmonellosis in neonatal calves rest upon demonstration of the causal organism in association with appropriate history, clinical signs and, to a lesser extent, pathological changes.

Occasionally, a syndrome involving dysentery and death in young calves has been found to occur as the result of infection with Clostridium perfringens, types B and C (Smith, 1962). However, Lozano and others (1971) have suggested that this situation is only rarely of importance. "Vibrionic" enteritis has also been described as an apparently rare event in young calves (Allsup and others, 1972), but is currently attracting increased interest (D.J. Taylor, personal communication).

(ii) Viruses

The gradual realisation that certain viruses may also be important as aetiological agents in neonatal calf diarrhoea has already been referred to. Probably the organism which has received the most attention has been rotavirus although others, in particular coronavirus and chlamydia, have also been studied in some detail.

A reovirus-like organism was isolated from calves examined during outbreaks of severe neonatal diarrhoea in Nebraska (USA) in the late 1960's (White and others, 1970). The organism was tentatively named "neonatal calf diarrhoea reovirus-like agent (NCDR)" and was later found to be widespread in North America (Mebus and others, 1973) and present in herds in several regions of England, Scotland and Northern Ireland (Woode and Bridger, 1975). The virus was later classed as a rotavirus and while morphologically similar to reoviruses it is now known not to be related to them serologically.

According to Mebus (1976) clinical signs in individual calves usually progressed in the following order: depression, anorexia, a few strings of thick saliva hanging from the lips and then diarrhoea. The onset of depression was rapid and in some cases calves progressed

from apparent normality to recumbency in two hours. The diarrhoeic period lasted five or six hours during which time the animal passed up to 300ml of liquid, yellow faeces, although the amount passed depended on the previous level of milk intake.

Material from field cases readily produced diarrhoea in susceptible calves when administered orally and in some cases, calves died even before diarrhoea occurred. In gnotobiotic calves, the incubation period was short (12.5 to 13.0 hours) but by 24 hours post-dosing, such a calf still appeared relatively normal, was still keen to suck and had only "pasty" faeces. However, if colostrum-deprived conventional calves were contaminated with E. coli during, or prior to, administration of rotavirus, 50 per cent died; on the other hand, most colostrum-deprived, conventional calves contaminated with E. coli after the rotavirus-induced diarrhoeic period survived. In gnotobiotic calves, rotavirus infection per se did not produce profuse diarrhoea, such a situation was characteristic of a combined rotavirus - E. coli infection and such calves rapidly became dehydrated (Mebus, 1975).

The sequential pathology of experimental infections in gnotobiotic calves was studied (Mebus, 1976). At both 30 minutes and four hours after the onset of diarrhoea, tissues appeared to be macroscopically normal. However, on histological examination of tissues obtained at the earlier time, the small intestine was found to be lined by tall columnar villous epithelial cells which showed fluorescent when stained specifically. Within four hours these cells were lost and replaced by low cuboidal cells.

Rotavirus infection may be confirmed by the demonstration of the agent in faeces or in small intestinal contents by either electromicroscopic examination or else by immunofluorescent staining

of faecal smears or infected cell cultures (Woode and Bridger, 1975). Herd infections may be demonstrated by the use of serum neutralisation tests on samples from either calves or adults (Mebus and others, 1973). However, the virus is known to be widespread in calves and other neonates (Woode and Bridger, 1975; Snodgrass and others, 1977) and has also been demonstrated in the faeces of apparently-normal calves so the mere finding of the organism is not without interpretive problems.

Coronaviruses have also been thought to play a part in the neonatal calf diarrhoea syndrome since the late 1960's (Stair and others, 1972) and again, this organism has been identified by the electromicroscopic examination of the faeces of diarrhoeic calves in Britain (Woode and others, 1974). The organism is similar to that which causes transmissible gastroenteritis (TGE) in pigs. Clinically, the effects in experimental calves differ from those of rotavirus infections in that diarrhoea lasts for up to six days with coronavirus infection and bacteriologically sterile calves have become moribund or died 48-62 hours after the onset of diarrhoea. Pathologically, lesions are more severe with greater dwarfing of villi and have been found in the colon and mesenteric lymph nodes (Mebus and others, 1973).

Another class of microorganism which has been associated with neonatal calf diarrhoea although not as yet on a large scale are the Chlamydiae. Bovine chlamydial strains were isolated from the faeces of young calves with diarrhoea and polyarthritis (Doughri and others, 1974). The calves became febrile and diarrhoeic within 24 hours of birth. Gross changes were present in the abomasum and throughout the intestinal tract although the terminal ileum was usually the most consistently and severely affected area; the lesions affected the mucosae and comprised oedema, congestion, petechial haemorrhage and ulceration.

Of the several other viruses or virus-like organisms which have been associated with neonatal calf diarrhoea, the virus of mucosal disease (MD, BVD) should perhaps be mentioned. While the true situation is unknown it is generally accepted that while this organism may sometimes infect and affect very young calves (Lambert and others, 1974) it is much more of a problem in older cattle.

(iii) Predisposing factors

Many of the widely recognised epidemiological features of neonatal calf diarrhoea can be ascribed to variations in the level of uptake of colostral globulins as the result of factors interfering with the natural formation of a dam-offspring bond and subsequent suckling (Selman, 1973). This situation has already been mentioned and will be discussed later, therefore the whole question of colostrum feeding will not be dealt with here. This section will deal only with those other factors which have been shown or considered as being causes of, or predisposing factors to, calf diarrhoea.

Several workers have emphasised their view that nutrition of the dam is of major importance. Evidence has been produced purporting to show that lower calf mortality rates occur where silage rather than hay forms the basis of the winter feeding regime (Withers, 1953) and much more specifically, another study (Stewart and McCallum, 1939) claimed to demonstrate that neonatal calf mortality rates were inversely correlated with the levels of Vitamin A in the milk being fed to the calves. Dam diet has also been associated with outbreaks of diarrhoea in suckling calves following changes to lush pasture (Blood and Henderson, 1974) although whether this situation is due to increased milk production or a toxin in the pasture was not known.

Overfeeding, irregular feeding and underfeeding of young calves have also been thought to act as predisposing factors (Amstutz, 1965). Such malpractices may well help to explain the "personal factors" which Withers (1952; 1953) felt to be so important and the calf-attendant-based mortality variations highlighted in Table 5. Other suggestions have included inefficient oesophageal groove mechanisms which allow milk to settle (and ferment) in the rumen (Comline and others, 1951) and inadequate secretory mechanisms in both abomasum and intestine (O'Connor, 1977). Claims have also been made that the quality of milk substitutes with special reference to the different techniques involved in the milk powder production systems may also significantly affect morbidity and mortality rates (Roy, 1959). In addition, much has been said of late regarding the fact that the feeding of "acid" milk substitutes is apparently associated with far lower m orbidity and mortality rates and significantly greater weight gains over the neonatal period (Low, 1978; 1979).

Fatigue, following prolonged transport, and rapid changes in ambient temperature have also been claimed as highly significant predisposing factors (Withers, 1952; 1953; Reisinger, 1965) along with such managemental factors as overcrowding and poor housing conditions (Reisinger, 1965; Acres, 1976). In addition, the dangers of continually using the same calf house and thus allowing infection build-up to occur, has long been recognised as a hazardous procedure (Roy and others, 1955; Roy, 1959; Reisinger, 1965; Mebus, 1976). Unfortunately, authorities (such as Inglis, 1960) who have produced blueprints for sound calf-rearing have consistently failed to produce data to support their claims that the systems they have advocated were in any way effective.

PATHOGENESIS

The following discussion will deal firstly with bacterial (i.e. E. coli), viral (i.e. rotavirus) infections and certain host factors. The other major determinant (i.e. immunity) will be dealt with in a separate section.

Colisepticaemia:

In most cases, the course of this disease is so rapid that diarrhoea is not often a presenting feature. Death usually occurs within a few hours and the strain of E. coli which is responsible may be cultured from the visceral organs, brain and joints in pure culture (Gay, 1965). Infection very probably gains access to the body via the lymphatics of the tonsils or through the umbilicus (Glanz and others, 1965).

In Gram-negative septicaemias, endotoxin is assumed to be released by dying bacteria directly into the systemic circulation (Zweifach and Janott, 1965) and this results in profound circulatory collapse and rapid death (Rosen, 1961).

In experimental endotoxic shock, a marked rise in haematocrit and plasma protein concentration occurs which is thought to be due to progressive haemoconcentration. In addition, there is a marked rise in blood glucose levels at around two to three hours post-injection (Tennant and others, 1973). Another response to parenteral endotoxin administration is the development of a leukopaenia. This has been shown to occur within five minutes of injection in young calves (Tennant and others, 1973). The largest percentage reduction was found to occur in the neutrophil fraction but in calves which survived for longer than ten hours, a secondary leukocytosis with neutrophilia developed. The same authors also found that this response was

dose-dependant and that relatively small doses produced a neutrophilia without a preceeding leukocytosis.

Surprisingly, little or no work appears to have been carried out to confirm unequivocally that the above changes occur in experimental colisepticaemia.

Enterotoxaemic colibacillosis:

In this form of colibacillosis, the development of diarrhoea is often rapid and death may occur within a few hours as the result of massive fluid and electrolyte loss (Gay, 1965). The problem arises as the result of infection by one of several strains of "mucoid" E. coli possessing A-type, K antigens (Gay, 1965) and their rapid proliferation in the small intestine. These strains of E. coli produce large quantities of enterotoxin(s) which, among their other properties, have the ability of affecting sodium and water transport through the intestinal epithelial cells (Gay, 1965; Bywater, 1970).

Enteropathogenic E. coli strains from young pigs and calves are able to produce cell-free substances which can cause dilation of ligated intestinal loops as the result of fluid accumulation (Bywater, 1970). Apparently, there are two distinct forms of enterotoxin activity. The first is heat-stable and is therefore referred to as "ST" (Smith and Halls, 1967) and the second is heat labile - the "LT" enterotoxin as originally described by Gyles and Barnum (1969). In the calf, enteropathogenic strains of E. coli can produce both forms of enterotoxin (Smith and Halls, 1967; Smith and Gyles, 1970).

It is generally accepted that ST and LT enterotoxins (and also cholera toxin) act only on the cells of the small intestine, not the colon. Diarrhoea due to infection by enterotoxaemic

(enterotoxigenic) strains of E. coli is due to the small intestine producing fluid and electrolyte secretions in such magnitude as to exceed the absorptive capacity of the colon (Leitch and Burrows, 1968). The host response evoked by LT and ST enterotoxins may differ in that while both can induce diarrhoea or dilatation of a ligated intestinal loop in susceptible experimental animals (Smith and Gyles, 1970), the action of ST is more rapid in onset but of shorter duration than that of LT (Stevens and others, 1972).

Enteric colibacillosis:

The problem of identifying this syndrome as actually being due, per se, to infection by non-enterotoxigenic strains of E. coli has already been stressed. This, coupled with the fact that a great amount of earlier work failed to define the type of diarrhoea being studied and the lack of a relevant "enteric colibacillosis" model in calves has lead to considerable confusion regarding the possible pathogenic mechanism involved - even supposing that such a condition does in fact exist. Nevertheless, many suggestions have been made which generally revolve around assumptions regarding loss of specialised (i.e. absorptive) cells from the small intestine, increased bowel motility, incomplete digestion, microbial multiplication and damage by, or absorption of, "toxins" (O'Connor, 1977). However, little experimental work appears to have been carried out to support such suggestions (i.e. in non-rotavirus, non-enterotoxaemic E. coli diarrhoea).

In contrast, the physiological and biochemical effects of (assumed or alleged) enteric colibacillosis have been studied in some detail. Most workers have reported a reduction, a loss of body fluids, a decrease in plasma sodium concentrations, an increase in blood urea nitrogen and a variable plasma potassium concentration

(McSherry and Grinyer, 1954). Clearly such changes will vary in degree in parallel with the severity of diarrhoea. Fisher (1971) emphasised the fact that even in calves which retained their milk intake, fluid loss from severe diarrhoea was likely to greatly exceed fluid intake. He also noted that when calves died within a few days of the onset of severe diarrhoea, haemoconcentration (as judged by haematocrit) was marked whereas in calves which suffered from a more long-standing syndrome were less likely to develop such marked changes in this parameter. In his opinion (Fisher, 1965) death from diarrhoea (i.e. in "enteric colibacillosis") occurred as the result of a combination of metabolic acidosis (the result of loss of Na^+ in faeces), severe dehydration and uraemia; the final cause of death was acute heart failure as the result of myocardial K^+ depletion (due to metabolic acidosis) and severe haemoconcentration.

Bacterial "adhesion" :

Another factor which is now considered to be important in the pathogenicity and pathogenesis of colibacillosis is the "stickability" of the various strains of *E. coli*. Strains of the organism which are pathogenic for the neonatal pig are usually distinguished from non-virulent strains by their ability to adhere to the intestinal epithelial cells, thereby overcoming removal by peristalsis (Gibbons, 1976). The K88 antigen on the surface of strains of *E. coli* which cause neonatal diarrhoea in pigs is an essential virulence determinant because its adhesive properties enable K88 positive strains to establish in the small intestine following attachment (Jones and Rutter, 1972). Once established, these strains multiply and synthesise enterotoxins thus leading to diarrhoea and even death (Smith and Halls, 1967). Another interesting feature of this phenomenon but as yet also undefined in terms of neonatal *calf*

diarrhoea, is that K88-positive E. coli do not attach to the intestinal epithelial cells of all piglets. At least two phenotypes occur which are the products of two alleles at a single locus which are inherited in a simple mendelian manner (Sellwood and others, 1974). A relatively simple laboratory test has been devised using K88-positive E. coli and brush borders prepared from epithelial cells of the pig small intestine and it has been possible to identify two pig populations (Gibbons, 1976). The majority of pigs tested have proved to be positive (i.e. adhesive); the minority of negative (i.e. non-adhesive) pigs exhibit this property at birth and throughout their life (Gibbons, 1976) and it has even been postulated that K88-positive bacteria colonise the gut of "adhesive" pigs more readily than "non-adhesive" pigs and hence the test may even indicate susceptibility to neonatal pig diarrhoea (Rutter and others, 1975). However, if "adhesive" pigs receive protective antibodies in mammary secretions they may appear to be resistant to infection. It has been suggested that this phenomenon might be exploited and thus allow for the selective breeding of diarrhoea-resistant pigs (Rutter and others, 1975).

Rotavirus:

Evidence that rotavirus is pathogenic for young calves is based on its ability to induce diarrhoea and pathological lesions in all non-immune calves challenged with both animal passaged and cell culture passaged virus, and the recovery of the virus from all such animals (Woode and Bridger, 1975).

Rotavirus is believed to infect primarily the epithelium of the small intestine. Immunofluorescent and histological studies (Mebus, 1971) on normal and infected calf intestine has suggested that diarrhoea arises as follows: after oral inoculation, the columnar

epithelial cells covering the distal two thirds of the villi of the upper small intestine become infected, and the infection rapidly progresses posteriorly. When the calf becomes depressed and diarrhoea begins, the villous epithelial cells from the upper small to the lower small intestine fluoresce, and there is an increase in fluid in the intestinal tract. As infection proceeds, infected epithelial cells move towards the tips of the villi, finally to be lost into the lumen. These cells are then replaced by cuboidal epithelial cells, but the migration of new epithelial cells from the base of the villi is not rapid enough to keep the tips of all villi covered. Gradually, the immunofluorescent epithelial cells are replaced by non-fluorescing cells and these latter cells are apparently resistant to infection because when calves were reinoculated with virulent virus 24 or more hours after having diarrhoea they remained normal (Mebus and others, 1971).

The primary lesion observed in calves infected with both rotavirus and coronavirus is very similar to lesions observed with transmissible gastro-enteritis (TGE) of pigs and it seems reasonable to suppose that the pathogenesis of these infections is similar. In TGE loss of function of the epithelial cells of the small intestine results in malabsorption, with diarrhoea and death as a consequence of ionic, bicarbonate and serum protein loss, acidosis and dehydration. Much of this effect is the result of the loss of the membrane bound enzyme lactase; undigested lactose then causes high osmotic pressure in the gut lumen (Woode and Bridger, 1975). Mebus (1975) has attempted to explain the role of bacteria in enteric viral infections as follows: organisms such as E. coli, frequently increase the severity of these viral infections, which alter normal digestion and absorption from the intestine. Therefore the altered physiology and

resultant increase in partially digested milk in the intestine enables bacterial proliferations to occur. Occasionally, damaged intestinal epithelium may even allow bacteria to enter the body. Although the primary infection may be viral, rapid dehydration and death within 12 to 24 hours after the onset of diarrhoea are due to bacteria.

IMMUNITY

The importance of colostrum in preventing neonatal calf diarrhoea was emphasised by Jensen (1893) although it seems likely that the subject was familiar to livestock owners long before that time. Smith and Little (1922) demonstrated the practical value of feeding colostrum to newborn calves on an experimental basis. The fact that calves do not receive significant immunity *in utero* was demonstrated by Smith (1930) who also recorded the presence of antibodies to what he considered to be diarrhoea-producing strains of *E. coli* in bovine colostrum. The same author also suggested that these were derived from maternal serum and that young calves were only able to absorb them from colostrum for a short time after birth.

The subsequent history of the gradual unfolding of this subject has been reviewed by Selman (1969). Very briefly, a wide variety of different techniques have been used to demonstrate that calves are only capable of absorbing macromolecules through their intestinal epithelial cells over the first 24 hours or so of life. The time-limit for the absorption of *immunologically adequate* amounts of colostral immunoglobulins (see below) is probably limited to the first few hours of life. It seems likely that the loss of this ability (intestinal shutdown) arises as the result of a gradual loss of a specific population of intestinal epithelial cells. Macromolecule absorption (it is a relatively non-specific phenomenon in young calves)

occurs only in the small intestine by a process known as micropinocytosis; once absorbed, the large molecules pass to the general circulation *via* the lacteals.

The overwhelming importance of colostral immunity was first demonstrated experimentally by Smith and Little (1922) and the protective factor in colostrum was shown to be in the acqueous (protein) fraction by Aschaffenburg and others (1949). The fact that not all colostrum-fed calves were capable of absorbing colostral immunoglobulin was first highlighted by Fey and Margadant (1961) and the enormous significance of individual variations in absorptive efficiency leading to markedly different post-colostral serum globulin concentrations and hence susceptibility to neonatal calf diarrhoea was first demonstrated by Gay and others (1965). Reasons for such variations were defined by Selman (1969) as follows:

(i) the age at which colostrum was first ingested,

(ii) the amount of globulin ingested and

(iii) the effect of presence of the dam.

The same author (Selman, 1969; 1973) was of the opinion that other epidemiological features of neonatal calf diarrhoea such as seasonality, place of birth, method by which colostrum was first presented merely reflected the probability of a calf's first having received colostrum by suckling its dam since this is by far the most efficient method of colostrum feeding (i.e. it usually leads to the absorption of maximal quantities of colostral immunoglobulin).

Other workers have explored the specific immunoglobulin classes present in bovine colostrum and have confirmed that the dominant immunoglobulin is IgG_1 (Penhale and others, 1970). This is

the immunoglobulin which is usually absorbed in greatest quantity and variations may well exist in the efficiency with which the different immunoglobulin classes are absorbed by newborn calves (Penhale and others, 1973). While more work certainly needs to be done regarding the subject of the protective roles of the various immunoglobulin classes, it would seem that IgM is mainly responsible for protection against systemic invasion of E. coli (i.e. colisepticaemia) (Penhale and others, 1970) and quite possibly IgG_1 is capable of protecting mucous surfaces (i.e. has an IgA-like role) until such time as the young calf is itself capable of producing IgA at about three weeks of age. However, it seems unlikely that immunologically adequate levels of IgG_1 will be secreted into the small intestine unless maximal serum concentrations are attained in the first instance.

Surprisingly little work has been carried out regarding the levels of specific colostral antibodies to known enteropathogenic strains of E. coli. However, it is known that protection against colisepticaemia is not strain-specific (Fey and Margadant, 1961). The value of circulatory (i.e. post-colostral) antibody against rotavirus is still somewhat controversial (Woode and others, 1975).

Other factors, such as the Vitamin A content of colostrum have been claimed as being of significant importance in protection against neonatal calf diarrhoea (Stewart and McCallum, 1938) but with increasing knowledge regarding immunological protection, together with the demonstration (Aschaffenburg and others, 1949) that the protective colostral factor lay in the acqueous rather than the fat fraction, this suggestion seems less and less likely (Selman, 1969).

TREATMENT

So few properly-controlled studies have been carried out regarding the treatment of neonatal calf diarrhoea that much of which has been written is of limited usefulness to the clinician. Similarly, lack of information regarding the nature of challenge and the immune status of affected calves makes interpretation difficult or impossible. In addition, many authors appear to be reluctant to quote other than anecdotal information and, frequently, one is left with the impression that their recommendations rarely if ever prove to be unsuccessful.

Therapeutic measures in neonatal calf diarrhoea have been reviewed by various authors (for example Fincher, 1956; Radostits, 1965; Blood and others, 1979) and in general terms, such measures revolve around the use of appropriate antibiotics, fluid and electrolyte therapy and other (supportive) procedures.

The review by Blood and others (1979) gives an extremely detailed and up-to-date account of the various treatments which may be administered to sick calves either alone or in combination. The authors emphasise the problems regarding the assessment of various claims regarding the efficacy of oral and/or parenteral antibiotics and also stress the problems regarding the choice of antimicrobial drugs to be used in an outbreak of neonatal calf diarrhoea, not least because there is often no time to carry out in-vitro sensitivity tests. Fluid and electrolyte therapy (including blood transfusions) are also discussed in some detail and the authors admit that the approach may be unsuccessful. In fact, modern opinion appears to oscillate between claims for high efficacy (Watt, 1965; 1967; Hamin and Hicks, 1975) to equally forceful claims (albeit under totally different circumstances) that the approach is valueless (de la Fuente, 1970).

Evidence from field studies (Selman - personal communication) would strongly suggest that the vast bulk of the 89,000 calves which die annually in Britain from one or other of the forms of neonatal calf disease have received some form of treatment, and in most cases this will have involved, at least, the administration of one or more antimicrobial preparations. This, together with the often random or haphazard use of such drugs and the problems associated with choice of treatments in neonatal calf diarrhoea (Radostits, 1965) strongly confirm the need for properly-defined and evaluated prevention methods and it is for this reason that the following chapter is devoted entirely to that subject.

CHAPTER 2

A REVIEW OF THE LITERATURE DEALING WITH THE PREVENTION OF NEONATAL CALF DIARRHOEA

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A REVIEW OF THE LITERATURE DEALING WITH THE PREVENTION OF NEONATAL CALF DIARRHOEA

Neonatal calf diarrhoea has long been recognised as a common, costly and complex problem but it is only relatively recently that some definite answers have been provided regarding various aetiological agents. During the last two decades, there has been a steady re-examination of many earlier hypotheses and a greater determination on the part of most research workers to justify and test their own aetiological theories experimentally. With improving technology it has been found possible to define several apparently different forms of neonatal calf disease (see Chapter 1) although, unfortunately, their differentiation still demands rather sophisticated laboratory expertise and equipment. For this reason attempts to establish the basic infectious cause(s) of field outbreaks are still only carried out on a very limited basis (i.e. in comparison with the scale of the problem) and this work is usually performed by teams of research investigators rather than by local "diagnostic" laboratories. To date, only a few attempts have been made to even construct a "league table" of infectious agents involved in outbreaks. Thus, most descriptions of neonatal calf diarrhoea have been made in ignorance of the agent(s) involved and the immune status (even in general terms) of the affected animal(s). In short, almost all of the work which has been documented on the treatment and prevention of neonatal calf diarrhoea has been based upon undefined (albeit apparently infectious) problems in calves which have only been assumed to have absorbed immunologically adequate amounts of colostral immunoglobulins.

It is essential to bear the above points in mind when considering the literature regarding measures which are advocated for the control of this very important problem. Other factors are of almost equal importance such as the wide variety of control measures which have been recommended over the years and the almost total lack of properly controlled studies which have been made to substantiate their value. Finally, it must be accepted that certain approaches to control (e.g. antibiotic and lactobacillus administration) may be viewed as either therapeutic or prophylactic depending on dose-rates and other factors. Indeed, in certain studies an examination of the protocols and conclusions do not give a clear indication whether the regime which has been advicated was preventing or merely minimising diarrhoea even when it did appear to have some beneficial effect.

This chapter will deal only with aspects of prophylaxis which have either received fairly widespread and detailed study or which seem to enjoy some measure of universal support and confidence. The review will be divided into four main sections, namely (i) husbandry factors (ii) immunological aspects (iii) the use of antibiotics and (iv) the use of Lactobacillus-containing compounds.

HUSBANDRY FACTORS

As is the case with many other infectious disorders, the prevention of neonatal calf diarrhoea largely revolves around increasing host resistance while at the same time decreasing pathogenic challenge However, certain aspects of the exercise do not readily fall into either category and others, perhaps, fall into both. Thus, calf comfort and a good diet may act by increasing the non-specific resistance (i.e. vigour) of a calf while at the same time, a properly-designed pen with a dry bed may also decrease the challenge of pathogens that a calf is

subjected to. The following section will deal with environmental considerations and diet.

(1) The environment

In attempting to summarise those factors which he considered to be of prime importance in preventing or minimising neonatal calf diarrhoea, Reisinger (1965) emphasised among other things, the value of an "..... intelligent application of known husbandry and veterinary principles and methods to ensure the birth of healthy calves into a favourable environment". In so doing, he was repeating what so many other authorities and advisors have advocated before albeit, unfortunately, in the absence of any definitive proof or actual (documented) support. In short, the general view would seem to be that newborn calves should be kept in "comfortable" accommodation, should be "well" fed and should also be subjected to as low a "pathogenic challenge" as possible.

The importance of high pathogenic challenge or infection "build-up" was first demonstrated experimentally by Aschaffenburg and others (1949). This group found that the incidence and occurrence of acute diarrhoea in calves under three weeks of age was directly proportional to the concentration of calves raised in close confinement and the length of time the calf house had been occupied by calves since the last clear-out, disinfection and vacancy. As this "occupation time" increases so does the incidence of diarrhoea and weight gains become significantly less (Roy, 1970). It has also been claimed that this effect may be magnified by the feeding of inferior milk replacers (Roy and Ternouth, 1972; Oxender and others, 1973). It seems likely that pathogenic challenge will increase markedly when some of the inhabitants of a calf house become diarrhoeic.

Clearly, in the light of this information it is imperative that periodic depopulation, clearing-out, disinfection and "resting" of the house, (if only for a few days) are practised whenever possible. However, it is equally obvious that such an approach is far easier for a calf-rearing enterprise such as a veal or intensive beef unit where large groups of similarly aged and size calves are admitted periodically. Attempts to evolve advisory systems for dairy farmers who lack purpose-built or properly-adapted calf accommodation and who also have a more sporadic calf supply system often prove to be far more difficult. Thus, Radostits (1974) advocated that dairy calves which were born indoors should be removed from their dams at birth to an individual calf pen which had been previously cleaned, disinfected and left vacant for at least a few days; he also stressed that such calves should not be penned together with older calves until at least two weeks of age. While it is obvious that such an approach may well decrease pathogenic challenge it is equally obvious if one accepts the view that such a procedure may well render calves inefficient absorbers of colostral immunoglobulins (Selman, 1969; Selman and others, 1971); therefore such a procedure may well result in the production of even more susceptible individuals. Clearly, it would be far better to consider both aspects of the problem and to ensure that all calves received colostrum in the most suitable way (see below) before being removed to the calf pens. Infection build-up within the same calf house through the winter months (in the face of a seasonal low absorption efficiency of colostral immunoglobulins) has been suggested as one of the major reasons for the very marked seasonal morbidity and mortality patterns seen in neonatal calf disease in Scottish dairy herds (Selman, 1973).

Little useful information is available regarding the true value of isolation of diarrhoeic calves from their cohorts but it

seems likely that such a practice will be of value to all concerned. Similarly, it would appear that in the face of an outbreak of calf diarrhoea, all calves which are still to be born should be reared (preferably in single pens) in a cleaned area which has not held young calves for at least some months.

The arguments which surround the relative merits of single versus multiple penning are considerable but are often decided by farmers and their advisors, not on the basis of disease control, but predominantly with regard to available labour, capital, space and feeding - techniques (Selman - personal communication).

(ii) Diet

Much of the earlier information regarding the importance of diet in the prevention of calf diarrhoea again rests upon unsubstantiated opinions and very often these opinions have been overtaken by newer management and feeding techniques.

Many earlier workers were of the view that the diet of pregnant cows very significantly affected the susceptibility of their offsprings to neonatal calf disease. The prevalence of diarrhoea in calves born to cows and heifers fed grass silage or artificially-dried grass was found to be much lower than that of calves born into hay and concentrate-feeding herds (Stewart and McCallum, 1938; Withers, 1953). However, the assumptions made in both studies have been questioned (Selman, 1969) and it has been emphasised by Sojka (1965) that vitamin (and especially vitamin A) supplementation of the diet of young calves has no significant effect upon the prevalence of diarrhoea.

Another factor which has been commonly claimed to affect the prevalence of calf diarrhoea is feeding technique. While admitting

that restricting milk intake did not always control calf diarrhoea, Blaxter and others (1952) strongly emphasised that in their view the overfeeding of young calves often led to an intestinal disturbance and diarrhoea. The same workers were also of the opinion that once-daily feeding often gave rise to diarrhoea due to overloading of the abomasum. Infrequent feeding was thought to give rise to diarrhoea by creating an intestinal disturbance which stimulated, or allowed, enteric bacteria to multiply (Lovell, 1959). Several workers have advocated three-times-daily feeding (for example Sheehy, 1949; Inglis, 1960). Dalton and others (1960) firmly believed that the main value of this practice was due to the fact that small frequent feeds simulated the more natural (suckling) state. However, this latter point is in some doubt since it has been shown, at least with suckling newborn dairy calves (Selman, 1969) that such individuals usually take large sporadic feeds. Finally, the feeding of very cold milk to young calves has been shown not to inevitably give rise to diarrhoea (Brownlee - cited by Selman, 1969).

As already stated, the above comments have to be considered with some degree of caution particularly in the light of relatively new (and often very successful) calf-rearing techniques such as once-daily feeding, six-days-per-week feeding, ad lib (machine) feeding of hot milk substitute and the ad lib (bin) feeding of cold milk substitute. However, there is some evidence to support the view that, at least in the past in Britain, certain milk substitutes were particularly dangerous (Roy, 1959).

That neonatal calf diarrhoea occurs more commonly in calves on a milk substitute diet than in those fed raw milk is a commonly expressed view although again there is little or no acceptable evidence

to support this view. However, distinct differences in susceptibility to diarrhoea have been shown to exist in calves fed different brands of milk powder. Observations and experiments on the effect of heat treatment of skimmed milk during the production of milk powder revealed that diets composed of powder which had been heated sufficiently to bring about denaturisation of a large proportion of whey proteins would, if given directly after the colostrum feeding period, predispose calves to an "E. coli localised intestinal infection" (Wood, 1955; Shillam and Roy, 1961). The heat treatment resulted in poor clotting of the milk by rennet (Shillam and Roy, 1963b) and a reduced digestibility of protein (Shillam and Roy, 1963a). Later work showed that the bucket-feeding of a "severely heat-treated milk" was associated with a reduced volume of abomasal outflow during the first hour after feeding, an increased output of undigested protein from the abomasum and a higher pH during the first six-and-a-half hours after feeding (Tagari and Roy, 1969). Such findings are also in accordance with the observed reduction in digestibility of protein and in the increased incidence of diarrhoea and mortality when calves were given "severely" heat-treated milk powder in an adverse microbiological environment (see above). It has also been suggested that diarrhoea is quite a common event in calves fed on a skimmed milk diet and that this is also due to the "excessive" protein contents of such diets (Roy, 1969).

IMMUNOLOGICAL ASPECTS

Some of the more important features of colostral immunoglobulin absorption and protection have already been dealt with in Chapter 1. The present section will deal with the more practical aspects of colostrum feeding, together with brief comments on the use of immune sera and vaccination.

(i) "Passive" protection from colostrum

Despite the various and numerous techniques which have been used to demonstrate the value of an early feed of colostrum there is still some measure of debate as to how important colostral protection really is. Again, many of the difficulties which have arisen are the result of worker's failing to record when and how colostrum was fed, the volume ingested, how efficient absorption was, the form of neonatal disease under consideration, whether or not specific protection was conferred and the degree (or method) of pathogenic challenge. One of the major misconceptions in this work has been that colostral immunoglobulin absorption is an inevitable sequel to the feeding of colostrum (Selman, 1973). As has already been noted, colostrum has to be ingested under certain critical, and now well-defined, circumstances in order that immunoglobulin absorption is maximal (Selman, 1969; 1973). Even then it must be accepted that while the mere feeding of colostrum is not a guarantee of survival, the obverse (i.e. colostrum-deprivation) usually means severe disease and even death under normal farming conditions. Moreover, there is now ample evidence to show that the bulk of colostrum-fed calves which die within the neonatal period (Leech and others, 1968) have in fact failed to absorb immunologically-adequate amounts of immunoglobulin (Gay and others, 1965; McEwan and others, 1970a; Selman and others, 1971).

The critical factors involved in ensuring that calves absorb maximal amounts of immunoglobulin would appear to revolve around the conditions surrounding the first feed of colostrum (Selman, 1969, 1973). Given optimum conditions, immunoglobulin absorption is maximal and under most practical circumstances the chances of a calf surviving the neonatal period are directly related to the serum concentration of passively-acquired immunoglobulin (Gay and others, 1965; McEwan and

others, 1970a). While it is not difficult to imagine how absorbed immunoglobulin protects young calves against invasive strains of E. coli for example (i.e. colisepticaemia) the situation regarding the way in which calves are protected against the effects of diarrhoea (McEwan, 1968) are as yet more difficult to explain. Nevertheless, this appears to be the situation; calves with high circulating levels of passively-acquired immunoglobulin may become diarrhoeic but do not suffer the deleterious fluid and electrolyte derangements which occur in diarrhoeic calves with lower serum concentrations (de la Fuente, 1970). It has been suggested by several workers that calves with high circulating levels of colostral immunoglobulin are able to survive enteric infection due to the large amounts of serum globulin which are re-excreted into the gut (McEwan, 1968).

The important factors associated with maximal immunoglobulin absorption are (i) time of feeding (ii) amount (mass) ingested and (iii) the presence of the dam (Selman, 1969). The timing of the first feed of colostrum is now accepted as being absolutely critical; intestinal "shutdown" starts at birth and proceeds so quickly that meaningful absorption is unlikely to occur after a calf has reached the age of ten hours. Thus, current advice is to feed as early as possible and certainly within six hours of birth. Since early suckling is not an inevitable consequence of loose-box or field-calving (Selman, 1969; 1973) it is essential to ensure that suckling has occurred within this time and not to assume that it has. The amount of immunoglobulin absorbed from an early feed of colostrum is directly dependent on the mass ingested (Selman, 1969) and since the concentration in colostrum is variable (Selman, 1969) it is essential to feed maximal volumes or, again, to ensure that calves suckle, or have suckled, to satiation. Ideally, calves should be encouraged to

ingest 5-7% of their bodyweight during their first suckling spell (Selman, 1969); in other words an average-sized (i.e. 35kg.) calf should be encouraged to consume 2.5 litres of colostrum. Such intakes though very large by normal farming standards, are quite comparable with those of naturally-suckling newborn dairy calves (Selman, 1969). The other important factor - as yet unexplained - is the fact that calves left with their dams are almost twice as efficient at absorbing immunoglobulin than non-mothered calves, irrespective of all other factors (Selman, 1969).

It has been suggested recently that to overcome some of the practical disadvantages involved in the above (ideal) advice and, in particular, the very close attention that is necessary during the early post-partum period, a large volume of colostrum might be administered by stomach-tube. Such a procedure is now known to allow significant absorption of immunoglobulin (Molla, 1978) and there is now increasing field evidence to suggest that it was a worthwhile part to play, especially on busier dairy farms (Selman - personal communication).

As already stated, the actual mechanism whereby absorbed colostral immunoglobulins exert their protective effects is still in doubt. At one stage it was firmly believed that such protection was strongly strain-specific and thus, even if a high-absorption calf was exposed to a strain of E. coli which its dam had never encountered, it was likely to succumb either to systemic invasion or a localised enteric infection. However, the surveys of Gay and others (1965) and McEwan and others (1970a) into the disease prevalence in calves of defined immunological status and gathered from a wide variety of sources would cast certain doubt on this hypothesis. In any case, the protagonists of strain-specificity rarely if ever attempted to confirm

that meaningful absorption had occurred in the calves under consideration. Moreover, strain-specificity has been shown by Fey and Margadant (1962) to be irrelevant at least in experimental colisepticaemia.

The protective role of colostrum under natural conditions in rotavirus infections is somewhat controversial. Woode and others (1975) showed that there was a rapid decline in the specific antibody content of colostrum within two days of calving, a situation which has been recognised for other specific antibodies and total immune lactoglobulin (Selman, 1969). Woode and others (1975) claimed that they were able to limit the rapid spread of diarrhoea among calves by orally administering colostrum with anti-rotavirus activity. Since they were of the view that high circulating specific antibody did not protect calves against rotavirus-induced diarrhoea they recommended the use of first-day (stored) colostrum as a prophylactic measure in outbreaks of "rotavirus diarrhoea". The same workers also recommended that it would be useful to check the antiviral activity of colostrum being deep-frozen for this purpose and even claimed that sour-stored (i.e. non-frozen) colostrum retained its antirotavirus activity. In practice, it seems unlikely that such monitoring of colostrum is readily feasible.

(ii) "Passive" protection with immune sera

Passive immunization induced by the parenteral administration of biological preparations has been the objective of a number of research programmes.

Aschaffenburg and others (1949) concluded that it was possible to prevent colisepticaemia with as little as 80ml of colostral whey

administered orally within the first few hours of life. Fey and others (1963) and Logan and Penhale (1971) also found that the intramuscular or intra-peritoneal administration of varying amounts of colostral whey proteins protected young calves against colisepticaemia but not neonatal diarrhoea. Attempts at prevention using serum, plasma and whole blood injections have yielded equivocal and often disappointing results (Gould, 1958; McDonald and Oakley, 1961; Watt, 1965). Similarly, Bosma (1968) observed that the intramuscular injection of two grams of bovine gammaglobulin shortly after birth affected neither their serum gamma globulin concentrations nor had any influence on the incidence and course of diarrhoea. In so doing, he demonstrated what many field workers have come to recognise when using commercially-available immune sera, namely that large amounts of gammaglobulin are necessary to bring about protection and, in general, this results only in protection against colisepticaemia, a problem which anyway, is easily prevented by the proper administration of colostrum. On the other hand, Urban (1968) reported good results in attempts to prevent neonatal calf diarrhoea by the parenteral administration of non-specific immunoglobulins at the rate of 1 ml/kg. bodyweight in colostrum-fed calves and 100-150 ml/kg. bodyweight in colostrum-deprived calves. Similarly, Logan and others (1974) claimed a partial reduction in mortality due to "enteric colibacillosis" following the parenteral administration of four grams of an IgM-rich fraction of bovine serum.

In short, no evidence has yet been produced to provide convincing support for the view that immune sera have a valuable part to play in the prevention of neonatal calf disease in comparison with the evidence which is available to testify to the value of properly-administered colostrum. Nevertheless, several commercial preparations are available in Britain.

(iii) The use of vaccines

Myers (1976) emphasised the difficulties of assessing the results of attempts to control neonatal calf diarrhoea by vaccination, not least because of the problems of diagnosing the specific cause of a particular outbreak or problem. Quite apart from making the results of recent trials almost impossible to interpret, the difficulties associated with differential diagnosis render much of the earlier studies impossible to assess quite simply because they were carried out at a time when E. coli was considered to be dominant, if not the sole, cause of neonatal calf diarrhoea. Nevertheless, many studies have been attempted and examples of the various approaches which have been made will be briefly dealt with under two headings: (i) colibacillosis and (ii) rotavirus infection.

Colibacillosis: With the above comments in mind (particularly in relation to earlier studies) the various approaches which have been attempted are three-fold, that is in-utero vaccination of the foetus, vaccination of the newborn calf and vaccination of the pregnant cow or heifer.

In a controlled experimental study, Gay (1971) demonstrated that it was possible to confer protection against challenge by invasive (i.e. colisepticaemic) strains of E. coli by vaccination of the foetus during the latter part of pregnancy. The vaccine used was a killed preparation. The study merely set out to examine the principle of in-utero vaccination and was in no way aimed at defining a practical approach to the problem. Similar studies, using an E. coli bacterin, revealed that such an approach not infrequently resulted in problems such as stillbirths and meningoencephalitis (Conne and others, 1973).

Other workers have studied the use of various preparations administered to newborn calves and both "immune" sera, vaccines and a combination of the two approaches have been tried.

Despite the fact that most calves which die of either colisepticaemia or neonatal diarrhoea do so before the age of two weeks (Withers, 1952) much interest has been directed towards evolving an effective vaccine against one or both problems. Fradkina (1935) reported that following vaccination of calves with "suitable organisms", a "considerable degree" of immunity was rapidly attained. Ulbrich (1954) concluded from his own studies that active immunization provided a useful prophylactic measure as long as the vaccine was prepared from a "herd-specific" strain of E. coli or from the most frequently isolated serotypes. Dam (1973) was of the opinion that a more rational approach to this type of control lay in the use of a serum preparation administered at birth, followed by vaccination. Another vaccinal approach was that reported by Porter and others (1975) which involved the oral administration (i.e. feed-incorporated) vaccine comprising heat-inactivated E. coli and Salmonella antigens. Improved health was registered in terms of a significant reduction in the incidence and duration of diarrhoea and in the need for antimicrobial therapy. Vaccinated calves also showed better weight gains and during the first three weeks of life the antigen-fed calves showed better faecal scores (i.e. less diarrhoea) together with a better appetite for concentrates. Such an approach obviously merits further consideration in view of the findings of the same group of workers when applying the same techniques to the problem of neonatal diarrhoea in pigs.

The concept of the specificity of colostral antibodies for certain pathogenic strains of E. coli, coupled with an increasing

awareness of the importance and mechanism of colostral protection, has prompted several groups of workers to investigate prevention by dam vaccination. Unfortunately (and quite apart from the problems of assessment outlined above) several of the studies have tended to assume that colostrum-feeding/suckling and colostral immunoglobulin absorption were almost inevitable under the conditions of their study.

Sellers and others (1962), using a heat-killed preparation of E. coli obtained from the faeces of six diarrhoeic calves from a single beef herd, also vaccinated pregnant cows and heifers. These workers failed to demonstrate any benefit whatsoever when the results were compared with a control group of calves whose dams were vaccinated with a non-pathogenic staphyllococcal (dead) preparation. Another attempt to control the disease (i.e. assumed colibacillosis) in this way was a large-scale experiment conducted by Shoenaers and others (1967) in Belgium. These workers claimed that their dam vaccination approach, using a killed preparation of supposedly pathogenic E. coli was effective although a subsequent analysis of their data carried out by Selman (1969) casts considerable doubt on this. More recently, Myers (1976) claimed significant success, using the dam-vaccination approach while at the same time emphasising the difficulties of obtaining and presenting convincing data.

Rotavirus infection: Oral vaccination, using an attenuated, cell cultured virus which is non-pathogenic for calves, has been claimed by Mebus and others, (1972) to be highly-effective in effecting a reduced incidence of rotavirus diarrhoea under field conditions. However, the validity of these claims has been questioned by Blood and others (1979) following a detailed appraisal of the available data. One of the most interesting facets to the whole question of the oral administration

of an attenuated rotavirus vaccine is precisely how the preparation works. It appears that a true immunity does not arise but that exposure and colonisation of the intestinal epithelium by the vaccine virus precludes invasion by virulent (i.e. wild) virus. An inactivated vaccine which is capable (following parenteral administration) of increasing the serum neutralising titre of cows by 20-40 fold has also been developed (Mebus and others, 1973a) and its use following dam vaccination is currently under assessment. The same workers have also developed a live, attenuated strain of calf coronavirus (Mebus and others, 1973b) for possible future use as an oral vaccine.

THE USE OF ANTIBIOTICS

All of the comments which have been made regarding the difficulties of interpreting the literature dealing with other methods of preventing neonatal calf diarrhoea, equally apply to the subject of antibiotic prophylaxis. Much of the reported work was carried out at a time when the problem was considered to arise as the result of a relatively simple bacterial infection and when the overwhelming importance of colostral immunity was not as widely appreciated as it is today. These factors, together with the apparent reluctance of many investigators to present other than anecdotal, un-controlled information, or even detailed information, makes this whole area of prophylaxis an extremely difficult one to assess. Current experience would seem to suggest that antibiotic prophylaxis per se (i.e. in agamma or hypogammaglobulinaemic calves subjected to high pathogenic challenge) is unlikely to significantly control a neonatal calf diarrhoea problem. Nevertheless, antibiotic prophylaxis apparently still remains as the method of choice with many farmers and veterinarians and a wide range of specifically-formulated antibiotic preparation are currently available in Britain.

Most of the earliest reports regarding the use of antibiotics in neonatal calf diarrhoea deal exclusively with their value as therapeutics. The results obtained, using such drugs as oxytetracycline (Katka, 1951; Pearson, 1954) appeared to be so impressive that it is not surprising that the use of these compounds and others in the prevention of this problem was soon advocated, particularly since increasing attention was being paid at that time to mass medication techniques in other livestock enterprises, particularly poultry. Thus, Pearson (1954) described the use of oral oxytetracycline, administered to calves on six problem farms during the first 48 hours of life as being totally effective in that no treated calves developed diarrhoea. Similarly, Roy (1959) advocated the use of oral chloramphenicol and parenteral oxytetracycline, both administered as soon as possible after birth. He did, though, infer that this was not necessarily effective in all cases.

However, in the first controlled study set up to investigate the relative efficacy of a number of different antibiotics, Dalton and others (1960) demonstrated that while differences existed between different compounds, none was completely effective. These workers did emphasise the importance of the route of administration and cited as an instance the failure of parenteral oxytetracycline to prevent neonatal calf diarrhoea, due, they suggested, to the fact that the drug was not excreted into the alimentary tract when given systemically. At about the same time, another worker (Glantz, 1962) emphasised the importance of choice of antibiotic in terms of the susceptibility of the various gut organisms; he concluded that it was important to choose a drug (for example, chloramphenicol) which would reduce the numbers of *E. coli* without upsetting the populations of other organisms such as the *Lactobacilli* and *Enterococci*.

One of the reasons for lack of efficacy of certain antibiotics in certain calves was considered by Dalton and others (1960) to be antibiotic resistance. If, as had been suggested by Smith and Crabb (1956), antibiotics were therapeutically effective in that they decrease the concentration of E. coli in the gut, then their failure to do so on some occasions could be explained by the presence of strains of the organism which were resistant to the particular antibiotic used.

Increasingly, the problem of bacterial resistance to antibiotics has preoccupied scientists although it should be emphasised that it is not a phenomenon exclusive to the family Enterobacteriacae. The prevalence of strains of E. coli resistant to such antibiotics as streptomycin, oxytetracycline and neomycin increased markedly between 1957 and 1963 (McKay and others, 1965) and Smith (1966) presented evidence to show that not only are many strains of E. coli capable of multiple resistance to a range of different antibiotics but also many such strains are often associated with severe disease in both man and his food animals. This situation has tempted clinicians to strongly recommend the use of in-vitro sensitivity tests prior to the commencement of treatment of neonatal calf diarrhoea (Radostits, 1974). On the other hand, Smith (1960) emphasised that, such was the rate at which resistant strains of E. coli could arise following the initiation of antibiotic therapy, the efficiency of antibiotics in eliminating sensitive strains was often masked by the extreme speed with which resistant strains could replace them. Transferable (infectious) resistance has been recognised as occurring commonly between members of the family Enterobacteriacae (Datta, 1962; Smith, 1966) and was stated by Walton (1968) as being the commonest form of acquired antibiotic resistance in this family of organisms.

Several groups of workers (for example, Dalton and others, 1960; Dalton, 1964; McKay and others, 1965; and others) have suggested that the failure of antibiotics to control outbreaks, or prolonged problems, of neonatal calf diarrhoea is likely to be due to infections with strains of bacteria resistant to the antibiotic which is being used. However, in the light of more recent information, the lack of knowledge regarding the immune status of the calves and the fact that E. coli was simply assumed to be the causal organism, means that so often such suggestions must be viewed with reservations. Nevertheless, the problems of antibiotic resistance in both human and veterinary medicine must not be under-emphasised (Swann, 1969) and as one step towards curtailing the problem, at least in Britain, there are now strict limitations on the choice of antibiotic which may be used as feed additives for either prophylactic or growth promotion purposes. The situation has also stimulated much interest in alternative, non-antibiotic methods of prevention for the enteric diseases of young food animals.

THE USE OF LACTOBACILLUS-CONTAINING PREPARATIONS

The search for effective, non-antibiotic methods for the prevention of neonatal diarrhoea in calves has led to a re-examination of certain approaches which were very probably cast aside during the early days of the "antibiotic era". One such approach was the use of lactobacillus-containing preparations.

Long before the existence of bacteria was recognised, the use of sour milk preparations such as yoghurt, kanmis and kefir was advocated for the treatment of gastro-intestinal disorders in both Europe and Asia (Hawley and others, 1959). The first systematic study of the use of sour milk in humans was by Metchnikoff (1910) who

was intrigued by the belief that Bulgarians who drank "bulgaricus" milk apparently lived longer than those who did not.

Early workers also studied the bacteriology of soured milk although many years elapsed before the problems associated with the implantation of certain of the organisms they found into the intestine (e.g. Lactobacilli) were appreciated (Hawley and others, 1959). The "lactic acid bacteria" was eventually shown to be either rod-shaped or spherical organisms which are now accepted as being in the genera Lactobacillus and Streptococcus respectively, of the family Lactobacteriacae. These bacteria are widely distributed in nature and may be isolated with relative ease from the mucosae of many different mammals and from milk fermenting food and green plants (Sandine and others, 1972). Unlike the Coli-aerogenes bacteria and the Enterococci, which are also considered to be an essential part of the normal gut flora, the intestinal Lactobacilli do not include any pathogenic organisms (Hawley and others, 1959). Under normal circumstances, the "lactic acid bacteria" rapidly colonise the intestine following nursing and suckling and under optimal conditions their presence within the alimentary tract appears to favour the establishment of a healthy, balanced flora. However, the mechanisms by which they exert these beneficial effects are still far from completely understood.

The phenomenon whereby the organisms in question help to prevent the establishment of pathogenic bacteria has been termed "bacterial antagonism" (Meszaros and Varga, 1975). This property is probably due, at least in part, to the ability of cultures of Lactobacilli (Wheater and others, 1951; Vincent and others, 1956) and Enterococci (Broch and others, 1963; Kafel and Ayres, 1969) to

produce antibacterial substances, the so-called "bacteriocines". Freeze-dried Lactobacilli (fed at the rate of 0.5 g/4 litres of milk daily) has been shown to lower the coliform quotient in calf faeces (Michna and Odovin, 1966) and Khausmann (1967) demonstrated that what he termed an antibiotic substance, produced by culturing L. acidophilus and certain gram-negative intestinal bacteria apparently had a therapeutic effect on diarrhoeic calves. Another probable benefit is that the organisms in question produce lactic acid, thereby decreasing the pH of the higher small intestine (Radulovic and others, 1976) and hence either restrict the multiplication of organisms such as E. coli or else limit their presence to the lower small intestine (Beslin, 1975). In view of these properties it is not surprising that a certain amount of interest has been paid over the years to the possible uses of these bacteria and/or their extracts for the prevention of various intestinal disease (Shaw and Muth, 1937; Kohler and Bohl, 1964; Reisinger, 1965).

Rettger and others (1935) summarised the conditions which they considered to be essential for the success of Lactobacillus therapy even before finite evidence existed to show its efficacy. They claimed that it was essential to use an intestinal strain, in their case L. acidophilus, and they also emphasised the importance of administering large numbers of viable cells, irrespective of whether "acidophilus" milk or a freeze-dried product was used. In the case of the former (liquid) preparation, it was found to be essential to store at low temperatures and to use as soon as possible; in the case of the freeze-dried preparation, factors such as the method of drying, the composition of the suspending medium and the method of reconstitution were all thought to be critical. Despite all of the above recommendations, little or no scientifically-acceptable evidence

was produced to support the claim that Lactobacillus therapy was beneficial in certain types of gastro-intestinal disease. However, some progress has now been made towards defining the value of this approach at least in young pigs and calves although, again, much of the work still suffers from many of the criticisms regarding valid control and diagnosis which have been already referred to on several occasions in respect of other prophyllactic procedures.

It has been shown that when young pigs were fed L. acidophilus, their growth rates were faster than controls on a non-supplemented diet and that the higher concentrations of lactic acid within their gut lumen apparently increased the absorption of calcium (Mollgaard, 1946). Increased weight gains were also noted by Olssen (1961) who fed young pigs 50g. of lyophilised L. acidophilus daily for ten days in an apparently successful attempt to counteract diarrhoea. Similar results, in terms of increased weight gains and a decreased tendency to diarrhoea in pigs fed L. acidophilus, have been noted in other, subsequent studies (Redmond and Moore, 1965; Nedyalkov and others, 1967). Hill and others (1970) also noted a reduction in both the severity and the duration of diarrhoea in L. acidophilus-fed pigs and suggested that this might be due to the demonstrable reduction in amine levels within the gut lumen of pigs on the supplemented diet.

Meszaros and Varga (1975) isolated Lactobacillus and Enteroccus strains from the faeces of 1-12 week old pigs and calves and "growing cattle" (sic). The isolates which were used experimentally were identified morphologically and biochemically according to the parameters established by Gibbs and Skinner (1966) as Bacillus bifidus, L. acidophilus and Streptococcus faecalis. Resistance was induced to bacitracin, neomycin and oxytetracycline

and pooled cultures of the bacteria were administered intranasally and orally to pigs between one and eight days of age. The results are summarised in Table 6 and it can be seen that there appeared to be a considerable reduction in diarrhoea and losses.

In some ways, the results of the work which has been carried out on the value of administering lactic acid bacteria to young calves are rather less convincing than those which have arisen out of the studies on young pigs. Early experiments carried out by Goudswaard (1968) to investigate the value of Lactobacilli and E. coli in calves infected with Salmonella dublin and Salm. typhimurium suggested that the approach had little to offer, at least in terms of Salmonellosis. Later studies by Thomas and others (1974) in which freeze-dried L. acidophilus was fed to calves and by Morrill and others (1977), who fed milk fermented by two species of Lactobacilli, were equally disappointing. On the other hand, studies carried out in Yugoslavia in which cultures of Lactobacilli were fed at birth (Radulovic and others, 1976) indicated that such a regime not only apparently prevented the occurrence of diarrhoea but also, when fed to control calves after its onset, acted therapeutically. Similarly, good results were obtained by Tournut and others (1976) in France following the administration of Lactobacilli and Enterococci immediately after birth and thereafter daily for the first three days of life.

Meszaros and Varga (1975) also studied the effect of feeding mixed cultures of specially prepared lactic acid bacteria to young calves (Table 7), although the numbers of experimental animals were considerably smaller than in their experiments involving young pigs. Once again, these workers demonstrated that very marked differences in morbidity and mortality could be ascribed to feeding such bacteria

	Total No. of suckling pigs at risk	Morbidity (diarrhoea)		Mortality	
		No.	%	No.	%
Treatment group	329	40	12.1	8	2.4
Control group	322	195	44.7	23	7.1

Table 6. The results of oral administration of various "lactic acid bacteria" to young, suckling pigs (from Meszaros and Varga, 1975).

	Total No. of young calves at risk	Morbidity (diarrhoea)		Mortality	
		No.	%	No.	%
Treatment group	40	5	12.5	0	0
Control group	35	19	51.4	9	25.1

Table 7. The results of oral administration of various "lactic acid bacteria" to young calves (from Meszaros and Varga, 1975).

under their experimental conditions. Somewhat similar studies, but using only a special strain of Streptococcus faecium (Milic and Sljivovacki, 1975) revealed relatively minor differences in terms of growth rates between test and control calves but unfortunately failed to subject their data to statistical scrutiny. The scale of differences revealed by their studies are summarised in Table 8.

The feeding of lyophilised L. acidophilus preparations is now a recognised procedure in certain countries, for example France, West Germany and Sweden. In many cases, it would seem that the approach is used therapeutically as well as prophyllactically. It is not yet available on a commercial scale in Britain and it seems very likely that much more work would need to be carried out in order to definitely establish its worth before its use became commonplace in this country. Nevertheless, it is an approach which appears to offer distinct advantages over the more usual preventive regimes (Beslin, 1975) and it is for this reason that the following studies were undertaken.

CHAPTER 3

INVESTIGATIONS INTO THE USE OF STREPTOCOCCUS FAECIUM (STRAIN 68) AS A PREVENTION AGAINST NEONATAL CALF DIARRHOEA

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INVESTIGATIONS INTO THE USE OF STREPTOCOCCUS FAECIUM (STRAIN 68)* AS A PREVENTION AGAINST NEONATAL CALF DIARRHOEA

Neonatal calf diarrhoea continues to be a major cause of loss to cattle farmers at least in the developed countries of the world. There is a clear need for effective prevention regimes and, increasingly, interest appears to be focussing on the development of methods which do not involve the mass medication of livestock with chemotherapeutics, many of which in any case, do not appear to be of proven value at least in the field of neonatal calf diarrhoea.

A considerable amount of work has been carried out, particularly over the last decade, into the prophylactic value of feeding lactic acid bacteria to young pigs and calves and, while much of the work can be criticised in terms of lack of standardisation or proper control, the results are such that further work should be undertaken in the hope that definite guidelines can be developed for calf rearers.

It was therefore decided to set up a series of controlled trials to investigate the efficacy of one commercial product which, although it has gained considerable support in several continental countries over the last few years, is as yet unavailable in Britain. There follows a detailed report of the five separate studies which were carried out.

(* LBC Concentrate M10, Bioferment SA, CH 6903 Lugano, Switzerland)

SECTION I: EXPERIMENT I: AN INVESTIGATION INTO THE EFFECT OF 150 mgm LBC, ADMINISTERED IN RAW MILK, TWICE DAILY FOR 21 DAYS.

In this first experiment, a dose-rate of LBC was chosen which appeared to be reasonable from earlier published data and a great deal of effort was made to standardise the type of experimental animal used, their immunological status and the (natural) pathogenic challenge to which they were exposed.

MATERIALS AND METHODS

Experimental calves

- Source and selection:

Thirty-three Ayrshire bull calves, approximately four to seven days of age, were purchased in two local cattle markets and admitted to the Department of Veterinary Medicine of the University of Glasgow Veterinary School on Monday and Tuesday, February 5 and 6, 1979.

On the afternoon of Tuesday, February 6, all of the calves were weighed and blood-sampled. Following a detailed clinical examination, thirteen of the 33 calves were rejected as being unsuitable for further study, one because it was much larger than the others, one because it was already diarrhoeic and 11 because they had extremely low zinc sulphate turbidity (ZST) values (see below).

The 20 calves which were selected for this first experiment were then ear-tagged, weighed, bled again and faecal swabs were taken. The mean body weight (20 calves) was 33.8 kg and the mean ZST value (20 calves) was 16.4 units.

The 20 selected calves were all housed in one room measuring 6m x 5m x 3m (high) and six pens were erected, each one measuring

2.0m x 1.65m ; these pens were arranged three on each side of a central feeding passage (see figure 1). Wooden slats of the "duckboard" type were placed on the floor of each pen and fresh straw was thrown on to these slats daily or more frequently if needed. None of the pens was cleaned out or disinfected during the 21 days experimental period. This room had not held young calves for some months and was cleaned, steam-washed and disinfected prior to the arrival of the experimental calves.

The calves were fed raw (i.e. non-pasteurised) cows milk which was obtained from the bulk tank of a nearby farm. The milk was fed warm and from a pail at the rate of approximately 5% of bodyweight, twice daily. No attempt was made to force unwilling calves to drink their allotted volume of milk although they were encouraged to do so. Different coloured plastic pails were used to feed the treatment and control groups. Hay was on offer by the beginning of the second week and calf pencils and water were available a few days later.

Experimental design

- allocation to groups:

The type of calf used in this study and their management was almost identical to that used in the much earlier studies of Dalton and others (1962) and Gay and others (1965). As the result of this latter work it became established that the most significant factor determining the survival of calves under these conditions was the level of passively-acquired colostral immunoglobulin as measured by the ZST test on admission. Consequently, great efforts were made to ensure that the two experimental groups of calves were of a similar immunological status. To that end calves with the same or similar ZST values were paired off and then allotted either to the treatment

FIGURE 1

ACCOMMODATION AND GROUPING OF CALVES

Experiment No. 1

High Treatment (3) Calves Mean body-weight on admission 31.5 Kg. (Range = 30.0-32.7 Kg) Mean ZST Value on admission 25.3 units (Range = 22-30 units)		Control Low (3) Calves Mean body-weight on admission 35.2 Kg. (Range = 33.6-36.3 Kg) Mean ZST Value on admission 9 units (Range = 7-12 units)
Intermediate Treatment (4) Calves Mean body-weight on admission 32 Kg. (Range = 28.1-36.3 Kg) Mean ZST Value on admission 15.5 units (Range = 12-20 units)	CENTRAL FEEDING PASSAGE	Control Intermediate (4) Calves Mean body-weight on admission 33.0 Kg. (Range = 29-39 Kg) Mean ZST Value on admission 15.7 units (Range = 12-20 units)
Low Treatment (3) Calves Mean body-weight on admission 37.2 Kg. (Range = 35.4-40 Kg) Mean ZST Value on admission 8.6 units (Range = 7-11 units)		Control High (3) Calves Mean body-weight on admission 35.1 Kg. (Range = 33.6-38.1 Kg) Mean ZST Value on admission 25 units (Range = 21-28 units)

DOOR

or control groups by the toss of a coin; the decision as to which group of ten calves became the treatment group was decided in the same way. The mean ZST value for the ten treatment calves was 16.4 units (range 7-30) and that for the ten controls 16.5 units (range 7-28). Thereafter, both groups were subdivided into high, intermediate and low ZST groups, that is relative to the range within each group. The various subgroups were then penned together as indicated in figure 1. The mean bodyweight of the treatment group was 33.4 kg and that of the controls 34.3 kg.

All of the above arrangements had been completed by the evening of Tuesday, February 6 and the trial commenced the following morning. At this stage all of the calves were judged to be healthy and the results of the faecal swabs later confirmed that none were excreting Salmonellae prior to the onset of the investigation.

- Dosing:

The preventive regime involved the treatment calves being dosed with a lyophilised concentrate of a single lactic acid bacterium, namely, Strep. faecium (Cernelle strain 68). This is available as LBC concentrate M10 (Bioferment SA, CH-6903 Lugano, Switzerland), one gram of which is stated to contain at least 35×10^9 viable organisms (see Appendix 1). Each treatment group calf received 150 mg of this preparation twice daily, mixed in milk; anorexic calves received their doses twice daily, mixed in a little distilled water. Dosing continued throughout the 21-day experimental period.

Experimental procedures

- Clinical examination:

Repeated (visual) examinations were carried out daily and at both feeding times but recordings were made only of the findings of

detailed examinations carried out once daily at about 09.00 hours. At this time, note was taken of the demeanour of the individual calves and their degree of activity (their ingestive behaviour was recorded at the appropriate time). Note was also made of rectal temperature, pulse rate, respiratory rate and the quantity and consistency of faeces. This latter observation was carried out either once defaecation had been stimulated following the insertion of a rectal thermometer into the rectum or else by an examination of an animals hindquarters, tail and perineum. A subjective assessment of the degree of diarrhoea was made by scoring the faeces as follows:

(i) normal faeces (0) - passed in small amounts and well-formed; sometimes covered with a film of mucous and yellowish brown in colour. On many occasions it was difficult or even impossible to stimulate defaecation in calves passing this type of stool.

(ii) diarrhoea (+) - passed in larger amounts but still holding form when falling on to bedding.

(iii) diarrhoea (++) - when stimulated, calves defaecated more readily and much of the faecal material drained into the straw; usually yellowish-green but sometimes creamy-white in colour. With this degree of diarrhoea there was usually a strong and offensive odour and faecal staining of the calf (and its neighbours) was common.

(iv) diarrhoea (+++) - these faeces were extremely fluid and were often discharged with explosive force, sometimes followed by straining; in recumbent calves, faeces would often dribble from their rectums. None was retained by the bedding and faecal staining of calves was commonplace; moreover, calves readily responded to stimulation with a rectal thermometer. In all cases the faeces smelled offensively. Usually the stools were yellowish-green and sometimes contained mucus although blood-clots were not commonly present.

When finally considering and expressing the results obtained, it should be emphasised that categories (0) and (+) were termed "normal" and (++) and (+++) "diarrhoeic".

- Post-mortem examination:

All calves which died were subjected to a pathological investigation but this was limited to a careful macroscopic examination.

- Sampling procedures:

As stated above, all calves were blood-sampled soon after admission. The blood samples were allowed to clot at room temperature and the zinc sulphate turbidity (ZST) was carried out on the serum so obtained according to the method of McEwan and others (1970).

Faecal swabs were removed soon after admission and thereafter once weekly or whenever calves became diarrhoeic. Bacteriological examinations were limited to a search for Salmonellae and were carried out by Dr. David Taylor of the Department of Veterinary Pathology, University of Glasgow Veterinary School. Salmonellae were not isolated from any calf, at any time.

All calves were weighed on admission and thereafter once weekly at 10.00 hours.

- Data collection and statistical analysis:

Individual case sheets were completed daily and a full data sheet (i.e. until death or for each of the 21 days of the experimental period for survivors) for each calf is presented in Appendices 3, 5, 7, 9 and 11.

Statistical analysis were carried out by Mr. Brian Wright, Chief Technician in the Department of Veterinary Medicine, University of Glasgow Veterinary School.

RESULTS

Morbidity

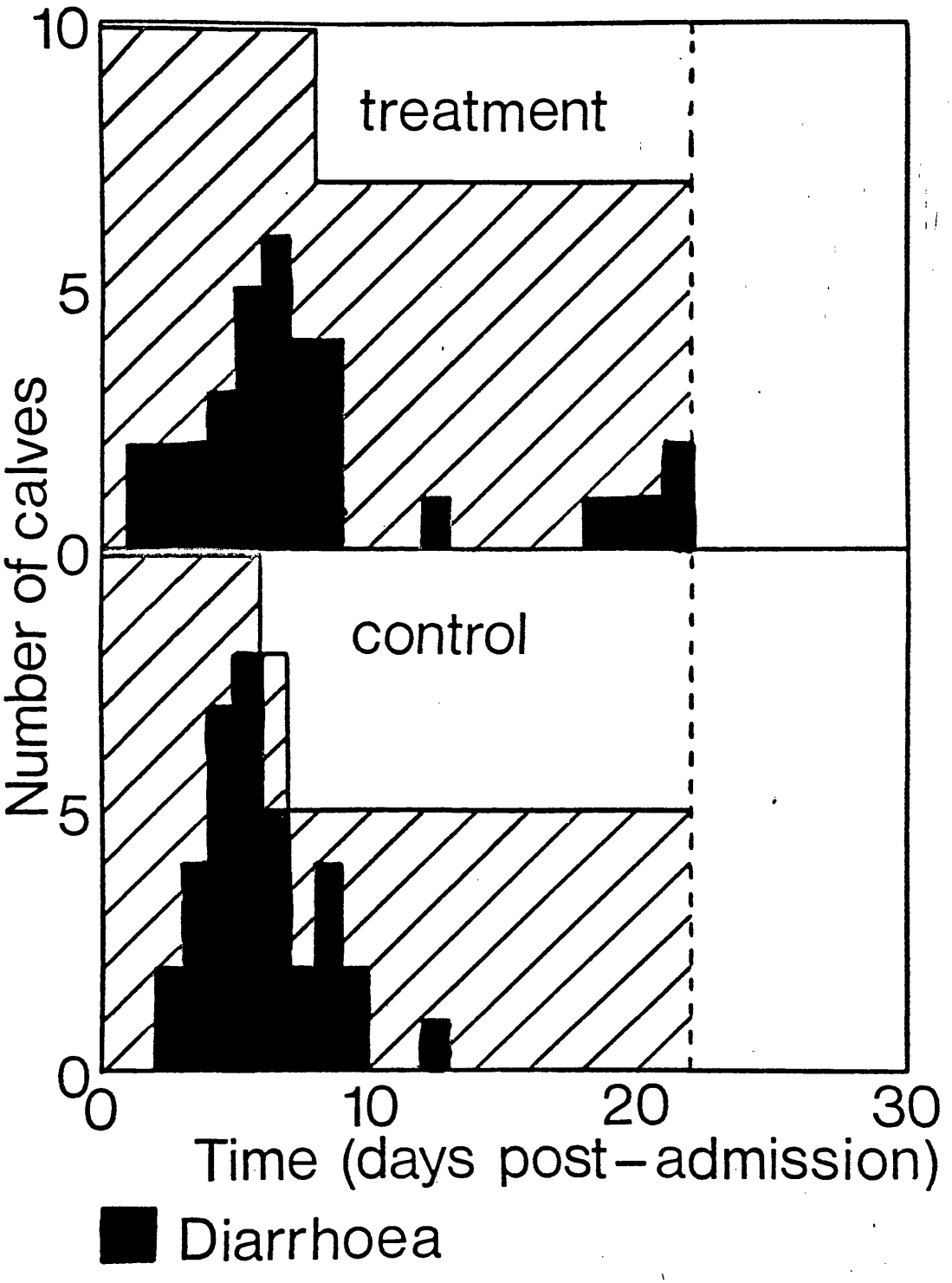
- Diarrhoea:

All calves save one developed diarrhoea at some point during the experimental period; the exception (10) was the heaviest calf in the trial, with an admission weight of 40 kg.

The prevalence of diarrhoea, as judged by the number of calves detected as being diarrhoeic at each daily clinical examination, is summarised in figure 2. It can be seen that in both groups of calves, the problem was at its height at around five or six days post-admission and at this stage there were no obvious differences between groups. Overall, the number of calf/diarrhoea days in each group was almost identical (treatments 34, controls 35) but in view of the different mortality patterns (see below) when calf/diarrhoea days were considered as a percentage of the total number of live calves at any one time the situation appeared somewhat different. Thus, the treatment group suffered diarrhoea on 34 of a possible 178 days (19%) whereas the control calves were diarrhoeic on 35 of 143 days (25%). Furthermore, it can be seen that in two of the treatment calves (2, 9) there was a second bout of diarrhoea between days 17 and 21 whereas no control calf was detected as being diarrhoeic after day 11.

In general, the condition of surviving calves in both groups improved markedly during the second week of the trial; they spent more time standing and grooming themselves and they were usually on their feet and bawling prior to feeding time. In addition, so few were diarrhoeic at this stage that there was less chance of calves defaecating over themselves and each other. However, with the onset of this second bout of diarrhoea, both diarrhoeic and non-diarrhoeic

FIGURE 2 : THE PREVALENCE AND PATTERN OF DIARRHOEA AND MORTALITY IN THE TWO GROUPS OF TEN CALVES STUDIED IN EXPERIMENT 1


10
5
0
treatment
control
5
0
0
10
20
30
Number of calves
Time (days post–admission)
Diarrhoea

treatment calves became dull, less interested in their food and in one case (2) recumbent.

During the earlier bout of diarrhoea, the duration in both groups averaged around four days. In most cases the colour and consistency were as described above for (++) and (+++) diarrhoea but one or two fatal cases had mild dysentery just prior to death.

The relationship between diarrhoea and ZST values on admission was not clearcut since all calves save one (10, ZST value 7) became diarrhoeic. However the two highest ZST treatment calves to die with values of 11 and 16 units were also found to have severe anterior lobe pneumonia; similarly, the highest ZST control calf to die (ZST value 21) was also very severely pneumonic. These were the only three calves to develop severe pneumonia and thus the ZST values of calves apparently dying from the effects of diarrhoea alone were, one treatment calf (11 units) and four controls (8, 12, 18 and 20 units).

- Respiratory signs:

Many calves in both groups developed a cough and nasal discharge during the second week of the trial although in only one animal (8) were gross respiratory signs exhibited.

- Conjunctivitis:

A bilateral, self-limiting conjunctivitis arose in most calves sometime during the first week of the trial. Some calves became quite severely affected although none progressed to corneal lesions and all recovered within a few days, irrespective of degree of severity.

- Umbilical infections:

Three calves (1, 3, 18) developed umbilical abscesses during the first week of the trial. These lesions quickly resolved following parenteral administration of procaine penicillin and streptomycin.

Mortality

In all, eight calves (40%) died and the mortality pattern is presented graphically in figure 2. Three treatment calves (30%) died, all of them on the eighth day of the trial. Five control calves (50%) died, two on day six and three on day seven. Comment has already been made regarding the relationship of the deaths to diarrhoea and also the ZST values on admission.

Growth rates

The individual and mean weekly weights for all of the calves in this trial are presented in Appendix 2. The mean weekly weight changes as a percentage of admission weights of both groups are presented in figure 3. It can be seen that while major differences were not present, there was a transient drop in the 14-day mean weights of the control calves against a steady increase in the treatment group. However, the mean weights at this stage (treatment calves, 41.4 ± 3.5 kg; control calves, 41.4 ± 1.4 kg) were identical.

The situation is somewhat different, however, on a consideration of mean weight gains over given periods. Thus, the mean 14-day weight gains (i.e. day 14 weight minus admission weight) are 7.4 ± 2.0 kg. (treatment calves) and 4.8 ± 1.5 kg. (controls). This difference is statistically significant ($p = 0.03$) although the corresponding values for days 7 and 21 are not significantly different. The weekly weight changes of individual calves are summarised in figure 4.

FIGURE 3 : THE MEAN WEEKLY BODYWEIGHT CHANGES (EXPRESSED AS A PERCENTAGE OF MEAN ADMISSION WEIGHTS) OF THE TWO GROUPS OF TEN CALVES STUDIED IN EXPERIMENT 1

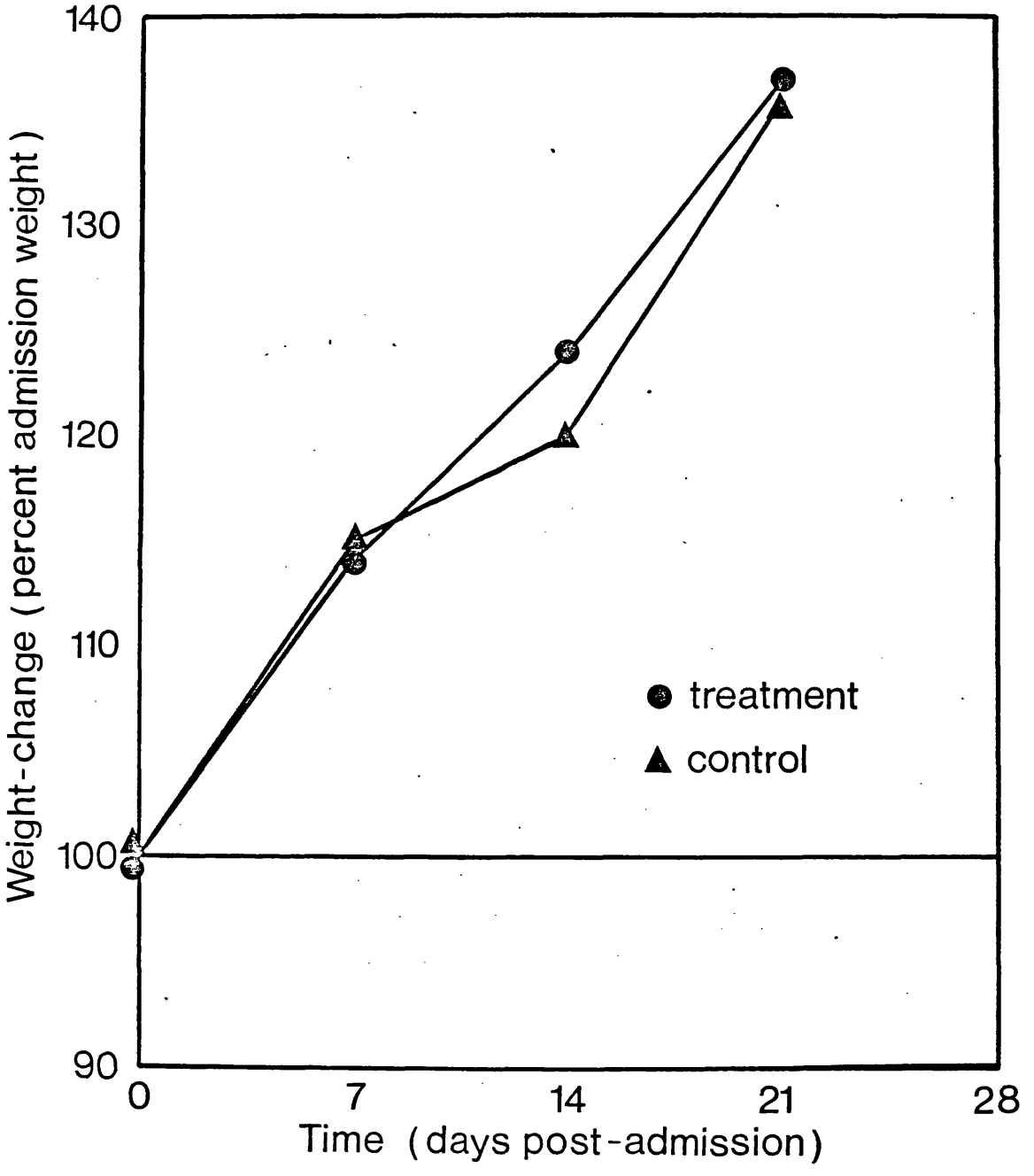
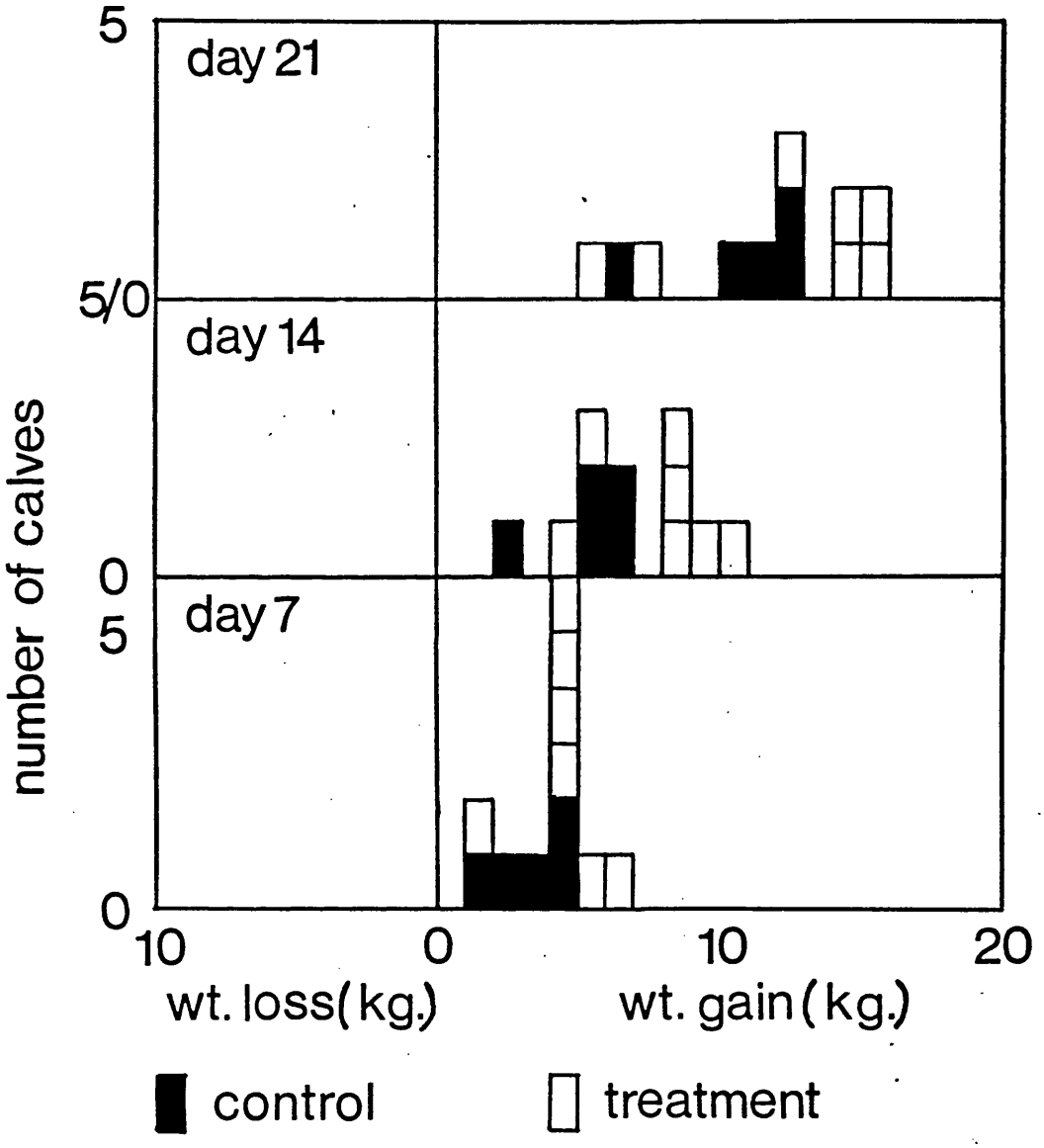

140
130
120
110
100
90
Weight-change (percent admission weight)
0
7
14
21
28
Time (days post-admission)
treatment
control

FIGURE 4 : WEEKLY WEIGHT CHANGES OF THE 20 INDIVIDUAL CALVES STUDIED IN EXPERIMENT 1


day 21
day 14
day 7
number of calves
5
5/0
0
5
0
10
0
10
20
wt. loss(kg.)
wt. gain(kg.)
control
treatment

DISCUSSION

In this experiment, a major effort was made to standardise the source, type and size of the calves, their immunological status (as measured by the ZST test), the conditions of management and feeding that they encountered once on test and the nature and level of a natural pathogenic challenge. No attempt was made to assess the level and nature of that challenge since it was felt that this in itself would have been a major exercise outwith the facilities available. Nevertheless, the syndrome encountered was apparently identical to that which has affected calves of this type in rearing experiments which have been carried out in these same buildings over the last two decades (Dalton and others, 1962; Gay and others, 1965; Selman - unpublished observations) and it was confirmed that Salmonella infections played no part in the problems which were encountered. It would seem that the experimental design was successful in that it encouraged a high and apparently "widespread" pathogenic challenge with an overall morbidity rate (i.e. diarrhoea rate) of 95% and an overall mortality rate of 40%. As in previous studies, carried out under broadly similar conditions (Gay and others, 1965; McEwan and others, 1971), deaths from uncomplicated diarrhoea occurred in calves with relatively lower ZST values.

The parameters which were used in an attempt to differentiate between the treatment and control groups of calves were, first, the level of disease (as judged by morbidity and mortality rates) and, second, growth rates.

Since diarrhoea affected all save one calf, the administration of LBC had no very obvious overall effect on the level of diarrhoea. However, the percentage of calf/diarrhoea days was higher in the

control calves (25% prevalence) than in the treatment calves (21% prevalence); moreover, in view of the fact that the 21% figure included a second episode of diarrhoea which affected only the treatment animals, the difference between groups during the first two weeks of the investigation were even more profound (i.e. 17% as opposed to 25%). The mortality rates in the two groups were also different; that for the control calves was 50% while the corresponding figure for the calves which received LBC was 30%.

An examination of the mean weekly weights of the two groups of calves revealed that a relative retardation in weight gains occurred in the control calves towards the end of the second week. However, this was found to be not statistically significant. On the other hand, a study of the mean weekly weight gains indicated that there were differences between groups, particularly on days 14 and 21. The difference between the control and treatment calves (i.e. 4.8 ± 1.5 kg and 7.4 ± 2.0 kg respectively) was found to be significant ($p = 0.03$) and it must be assumed that this difference was due to difference in the prevalence of diarrhoea between the two groups.

The finding that only one statistically significant difference was demonstrable between the two groups of calves must be viewed in the light of the overall results. Thus, there was more diarrhoea in the control calves and, at least at one stage, a decreased growth rate. In addition, there was a higher mortality rate (i.e. from diarrhoea or diarrhoea and pneumonia) in the untreated animals.

These results indicate that LBC, dosed at the rate of 150 mgm twice daily for 21 days has a markedly beneficial effect in terms of health, performance and survival, at least through the neonatal period.

The extremely high morbidity and mortality in both groups of calves is an indication of the harsh nature of this experimental model and it is quite conceivable that the beneficial effects of LBC administration might be even more dramatic under better conditions of husbandry and housing and with a better "type" of calf. However, the second wave of dullness and/or diarrhoea which, although relatively mild, was limited to the treatment calves should sound a note of warning in that it is just possible that the dosage rate and regime used, over a three-week period, might have been associated with undesirable side-effects.

SECTION 2: EXPERIMENT 2: AN INVESTIGATION INTO THE EFFECT OF 75 mgm LBC ADMINISTERED IN RAW MILK TWICE DAILY FOR 14 DAYS.

In view of the appearance of diarrhoea and/or dullness in some of the surviving treatment calves towards the end of the experimental period in the previous trial, a new investigation was instigated using LBC at a dose rate of 75 mgm twice daily for 14 days.

MATERIALS AND METHODS

Experimental calves

The methods of acquisition and selection of the calves used in this experiment was as outlined in Section 1. Twenty-five calves were purchased from local markets and admitted to the Department on Monday and Tuesday, March 19 and 20, 1979. All were Ayrshire bull calves aged between four and seven days on arrival. Five were rejected as unsuitable, one because it was ill and four which were agamma or markedly hypogammaglobulinaemic. The mean bodyweight of the 20 selected calves was 37.7 kg and the mean ZST value was 17.1 units. Sampling procedures were as already described and the calves were grouped and allotted to pens in the same room as was used in experiment 1. This room had been cleared out, but not disinfected, after the surviving experiment 1 calves were moved out, 28 days after admission. The lay-out of the housing and the distribution of calves is summarised in figure 5. All other management and feeding systems were as in experiment 1 except that the LBC dose-rate was 75 mgm twice daily and the trial (which commenced on Wednesday, March 21) lasted only 14 days. The mean bodyweight of the ten treatment calves was 37.7 kg (range 33-45) and of the ten controls 37.7 kg (range 33-44). The mean ZST value was 17.1 units (range 5-32) in the treatment calves and 17.1 units

FIGURE 5

ACCOMMODATION AND GROUPING OF CALVES

Experiment 2

High Treatment (3) Calves Mean body-weight on admission 37 Kg. (Range = 35-40 Kg) Mean ZST Value on admission 29 units (Range = 26-32 units)			Control Low (3) Calves Mean body-weight on admission 36.3 Kg. (Range = 33-40 Kg) Mean ZST Value on admission 8 units (Range = 7-9 units)
Intermediate Treatment (4) Calves Mean body-weight on admission 38.5 Kg. (Range = 36-44 Kg) Mean ZST Value on admission 16 units (Range = 11-22 units)		CENTRAL FEEDING PASSAGE	Control Intermediate (4) Calves Mean body-weight on admission 37.2 Kg. (Range = 34-42 Kg) Mean ZST Value on admission 13 units (Range = 9-18 units)
Low Treatment (3) Calves Mean body-weight on admission 37.3 Kg. (Range = 33-45 Kg) Mean ZST Value on admission 6.6 units (Range = 5-8 units)			Control High (3) Calves Mean body-weight on admission 39.6 Kg. (Range = 35-44 Kg) Mean ZST Value on admission 31.6 units (Range = 22-38 units)

DOOR

(range 7-38) in the controls. Salmonellae were not found in any faecal sample removed from any calf during the 14-day experimental period.

RESULTS

Morbidity

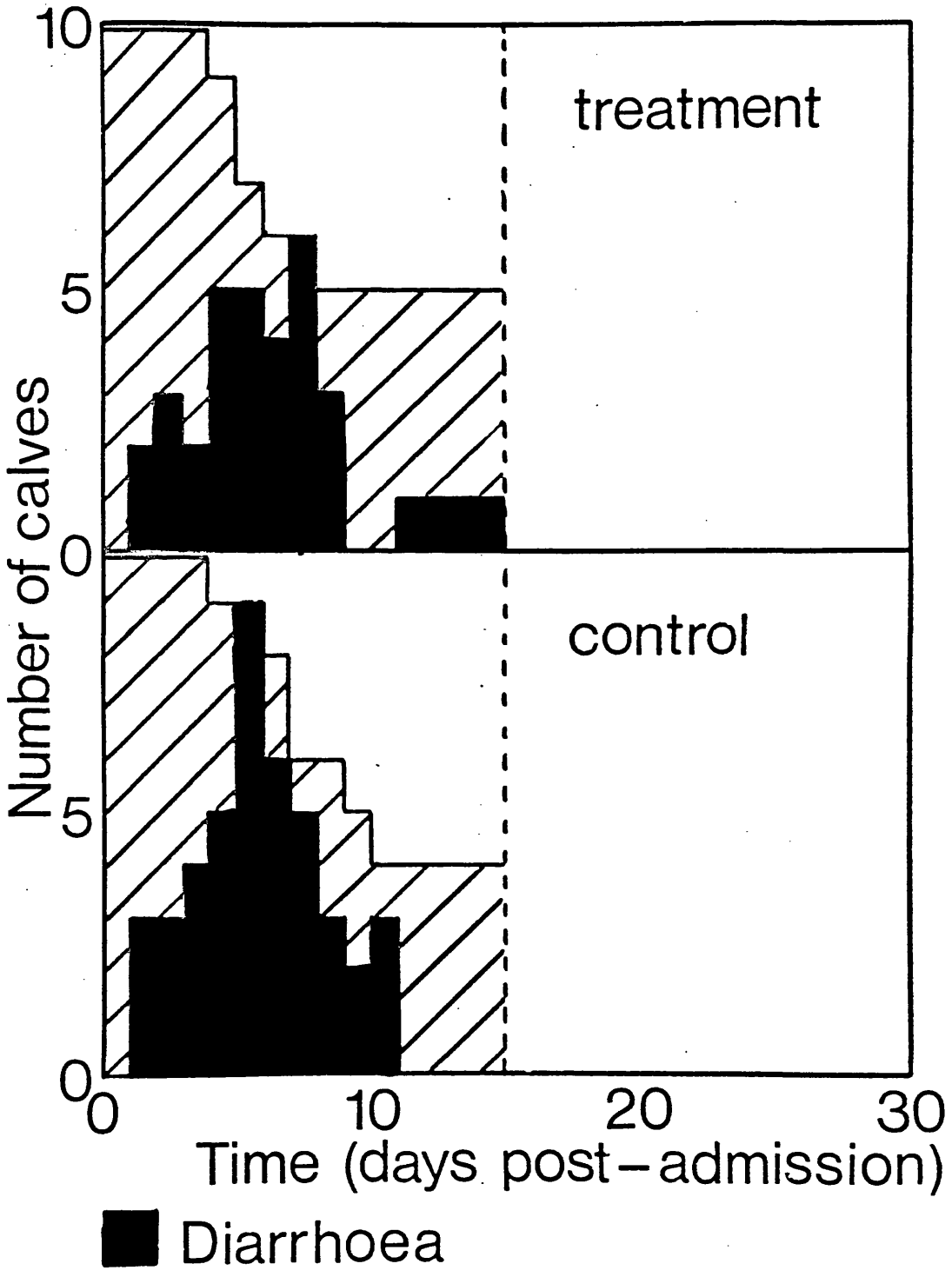
- Diarrhoea:

Sooner or later all calves in both groups became severely diarrhoeic and the peak prevalence (see figure 6) was on the fourth day of the trial in the control calves and on the sixth day in the treatment group. No obvious difference existed between the two groups at this stage, but the total number of calf diarrhoea days when expressed as a percentage of live calf/days (i.e. taking into account the different mortality pattern between the two groups) reveals that the figure for treatment calves was 34 out of a possible 98 days (35%) and for the controls 44 out of a possible 99 days (44%). Despite the fact that this trial only lasted 14 days, it can be seen from figure 6 that diarrhoea persisted over a longer period in some of the surviving treatment calves than in the controls.

During the first week of the trial almost all of the calves were extremely dull and many were disinterested in feeding and failed to clean themselves. However, as the second week progressed the survivors in both groups gradually brightened with the exception of one calf (28) which remained diarrhoeic throughout most of the second week.

Once again, a clearcut relationship between the occurrence and ZST values was impossible to define since all calves became diarrhoeic. However, in the calves which were considered to have died predominantly from the effects of diarrhoea, the mean ZST value

FIGURE 6 : THE PREVALENCE AND PATTERN OF DIARRHOEA AND MORTALITY IN THE TWO GROUPS OF TEN CALVES STUDIED IN EXPERIMENT 2


10
5
0
treatment
control
Number of calves
0
10
20
30
Time (days post–admission)
Diarrhoea

was 16 units (range 5-35) while the mean value for calves which died as the result of diarrhoea and respiratory disease were 25 units (range 7-38).

- Respiratory signs:

Several calves in both groups (21, 24, 31, 40) developed respiratory signs, severe depression and fever during the first week of the trial. All died and all were found to have severe anterior lobe pneumonia of the exudative type. Several other calves were coughing occasionally at the termination of the study.

- Conjunctivitis:

Only one calf (25) developed transient, bilateral conjunctivitis.

Mortality

A total of 11 calves (55%) died during the 14 day experimental period; five of these (50%) were in the treatment group and six (60%) were control animals. The pattern of mortality is summarised in figure 6. Comment has already been made regarding the relationship between diarrhoea, respiratory disease, mortality and the ZST values on admission.

An interesting post-mortem finding was that in four of the five treatment calves and in five of the six controls, there was either severe abomasal congestion (two cases) or else definite erosions (seven cases). The cause of this was not determined, although it should be emphasised that this was likely to have been a terminal event in that only one fatal case was seen to have slightly blood-tinged faeces just prior to death.

FIGURE 7 : THE MEAN WEEKLY BODYWEIGHT CHANGES (EXPRESSED AS A PERCENTAGE OF MEAN ADMISSION WEIGHTS) OF THE TWO GROUPS OF TEN CALVES STUDIED IN EXPERIMENT 2

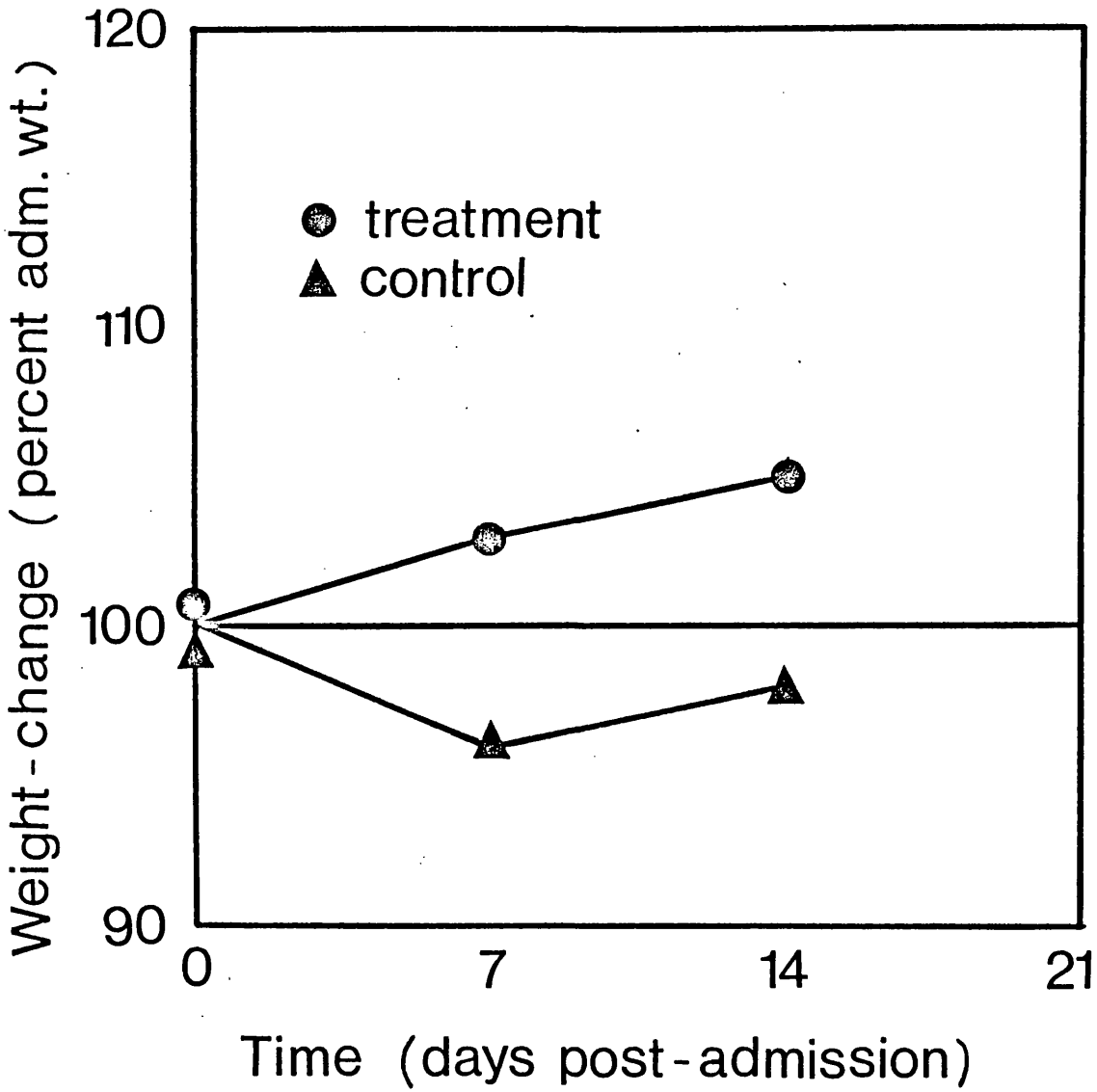
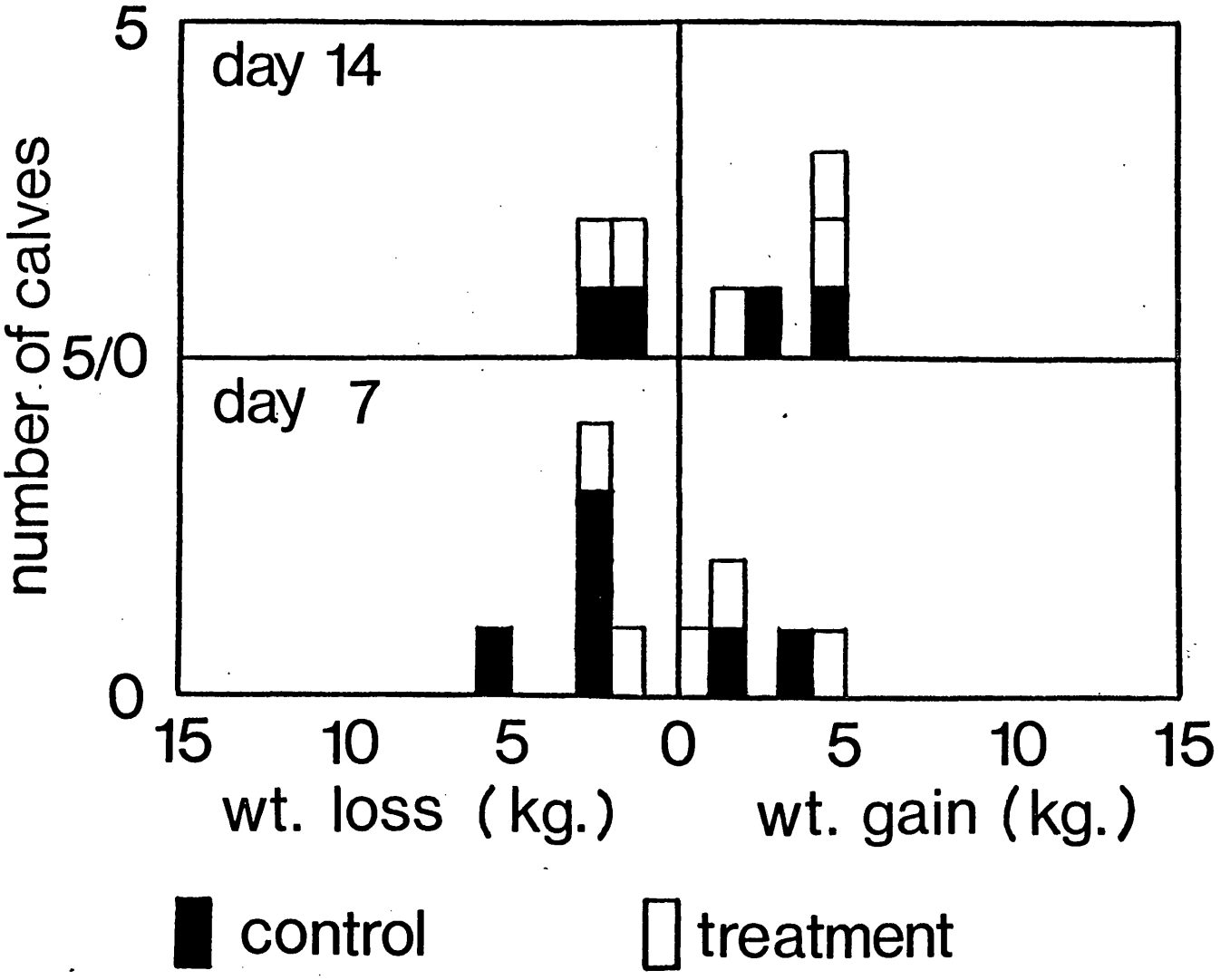

120
110
100
90
Weight-change (percent adm. wt.)
treatment
control
0
7
14
21
Time (days post-admission)

FIGURE 8 : WEEKLY WEIGHT CHANGES OF THE 20 INDIVIDUAL CALVES STUDIED IN EXPERIMENT 2


5
day 14
number of calves
5/0
day 7
0
15
10
5
0
5
10
15
wt. loss (kg.)
wt. gain (kg.)
control
treatment

Growth rates

The individual and mean growth rates for the calves in this experiment are presented in Appendix 4. The mean weekly weight changes as a percentage of admission weights of both groups are presented in figure 7. It can be seen that distinct differences were present between the two groups of calves on both the seventh and the fourteenth day of the trial despite the fact that the mean bodyweights at the commencement of the trial were identical. The mean values were as follows: on day seven, treatment calves 38.8 $\pm$ 3.2 kg, controls 36.2 $\pm$ 4.2 kg; on day fourteen, treatment calves 39.6 $\pm$ 4.8kg controls 37.0 $\pm$ 2.6 kg. However, the differences were not found to be statistically significant.

The weight changes on days seven and fourteen of the individual calves are summarised in figure 8 from which it can be seen that approximately equal numbers of calves in both groups had actually lost weight on each occasion. The mean weight gains by day 14 were 1.2 $\pm$ 2.5 kg (treatment calves) and 0.75 $\pm$ 2.4 kg (controls) but, again, this difference was not statistically significant.

DISCUSSION

This investigation differed in three ways from the previous experiment. First, no attempt was made to disinfect and rest the calf accommodation after the departure of the survivors from experiment 1 (the pens were merely cleaned out); second, the dose-rate of LBC was halved (i.e. 75 mgm twice daily for the duration of the experiment) and the trial lasted only 14 days. The likely increase in pathogenic challenge which was anticipated as a result of introducing the calves into more contaminated conditions appears to have resulted in the higher morbidity and mortality rates.

"Acute undifferentiated diarrhoea" (Blood and others, 1979) occurred at the 100% level but was more prevalent during the experimental period in the untreated calves (i.e. 44% calf/diarrhoea days compared with 35% in the treated animals). Again, when deaths due to diarrhoea alone were considered, it appeared that there was a relationship between this and ZST values on admission, although this was less distinct than in experiment 1. The overall mortality rate was 55% (50% in the treated calves, 60% in the controls). As in experiment 1, deaths were due to either diarrhoea per se or else diarrhoea and pneumonia and these occurred between days four and ten.

A comparison between the mean weekly weights of the two groups of calves revealed higher weekly mean weights in the treated animals but these differences were not significant. The treated calves also had higher weight gains (e.g. at 14 days, treatment calves, 1.2 ± 2.5 kg, controls 0.75 ± 2.4 kg) but, again, these differences were statistically insignificant. It is possible that these effects, at least in part, were due to the high mortality rates and the consequent rapidly diminishing group numbers although note should also be made of the fact that, in contrast to the results of experiment 1, a number of calves in both groups actually lost weight during the trial.

In short, these results revealed a somewhat similar situation to that found in experiment 1. Despite what was very probably a higher pathogenic challenge, LBC (even at a halved dose-rate) appeared to reduce the severity and/or duration of diarrhoea and also mortality rates. The performance (as measured by weight changes) was also greater in the treated calves, presumably as the result of a decreased tendency to, or severity of, diarrhoea. Individual assessment and analyses of morbidity, mortality and growth-rates did not produce significant

differences but when viewed together, these inter-related events must surely have been of some significance and again, the effects of LBC are likely to have been more obvious in a less harsh management system.

SECTION 3: EXPERIMENT 3: AN INVESTIGATION INTO THE EFFECT OF 150mgm LBC ADMINISTERED IN RAW MILK TWICE DAILY FOR SEVEN DAYS FOLLOWED BY 75mgm LBC ADMINISTERED IN RAW MILK TWICE DAILY FOR A FURTHER SEVEN DAYS

In the light of the two previous experiments, it was decided to investigate the value of dosing LBC at the rate of 150 mgm twice daily for seven days, followed by 75 mgm twice daily for a further seven days.

MATERIALS AND METHODS

Once again, the methods of acquisition, selection and standardisation of the two groups of calves and their management and feeding were as indicated in previous experiments.

On this occasion, 23 calves were purchased and three were rejected on the grounds of ill-health prior to the onset of the trial on Thursday, April 18, 1979. The 20 calves which were selected for further study had a mean bodyweight of 36.7 kg and a mean ZST value of 9 units. It should be emphasised that this very low ZST value was the result of an extremely low set of values in the 23 selected calves and hence represented lack of choice rather than an attempt to study the effect of LBC in calves with particularly low ZST values. Repeated examination of faecal swabs failed to confirm the presence of *Salmonellae*.

The calves were accommodated in the same room as previously and the housing and penning arrangements were as set out in figure 9. The accommodation had been cleared of the experiment 2 calves approximately one week prior to the start of this experiment; the pens had been cleaned out but not disinfected prior to the introduction of the experiment 3 animals.

FIGURE 9

ACCOMMODATION AND GROUPING OF CALVES

Experiment 3

High Treatment (3) Calves Mean body-weight on admission 37 Kg. (Range = 35-41 Kg) Mean ZST Value on admission 16 units (Range = 11-22 units)		Control Low (3) Calves Mean body-weight on admission 38.6 Kg. (Range = 37-41 Kg) Mean ZST Value on admission 2.6 units (Range = 2-3 units)
Intermediate Treatment (4) Calves Mean body-weight on admission 34.5 Kg. (Range = 33-37 Kg) Mean ZST Value on admission 8.7 units (Range = 7-10 units)	CENTRAL FEEDING PASSAGE	Control Intermediate (4) Calves Mean body-weight on admission 39.7 Kg. (Range = 31-46 Kg) Mean ZST Value on admission 8.2 units (Range = 4-11 units)
Low Treatment (3) Calves Mean body-weight on admission 37.6 Kg. (Range = 32-41 Kg) Mean ZST Value on admission 2.3 units (Range = 2-3 units)		Control High (3) Calves Mean body-weight on admission 32.3 Kg. (Range = 26-41 Kg) Mean ZST Value on admission 16.3 units (Range = 11-24 units)

DOOR

The treatment calves were dosed with LBC in their milk at the rate of 150 mgm twice daily for the first week and 75 mgm twice daily for the second week of the experiment. The total experimental period was 14 days.

RESULTS

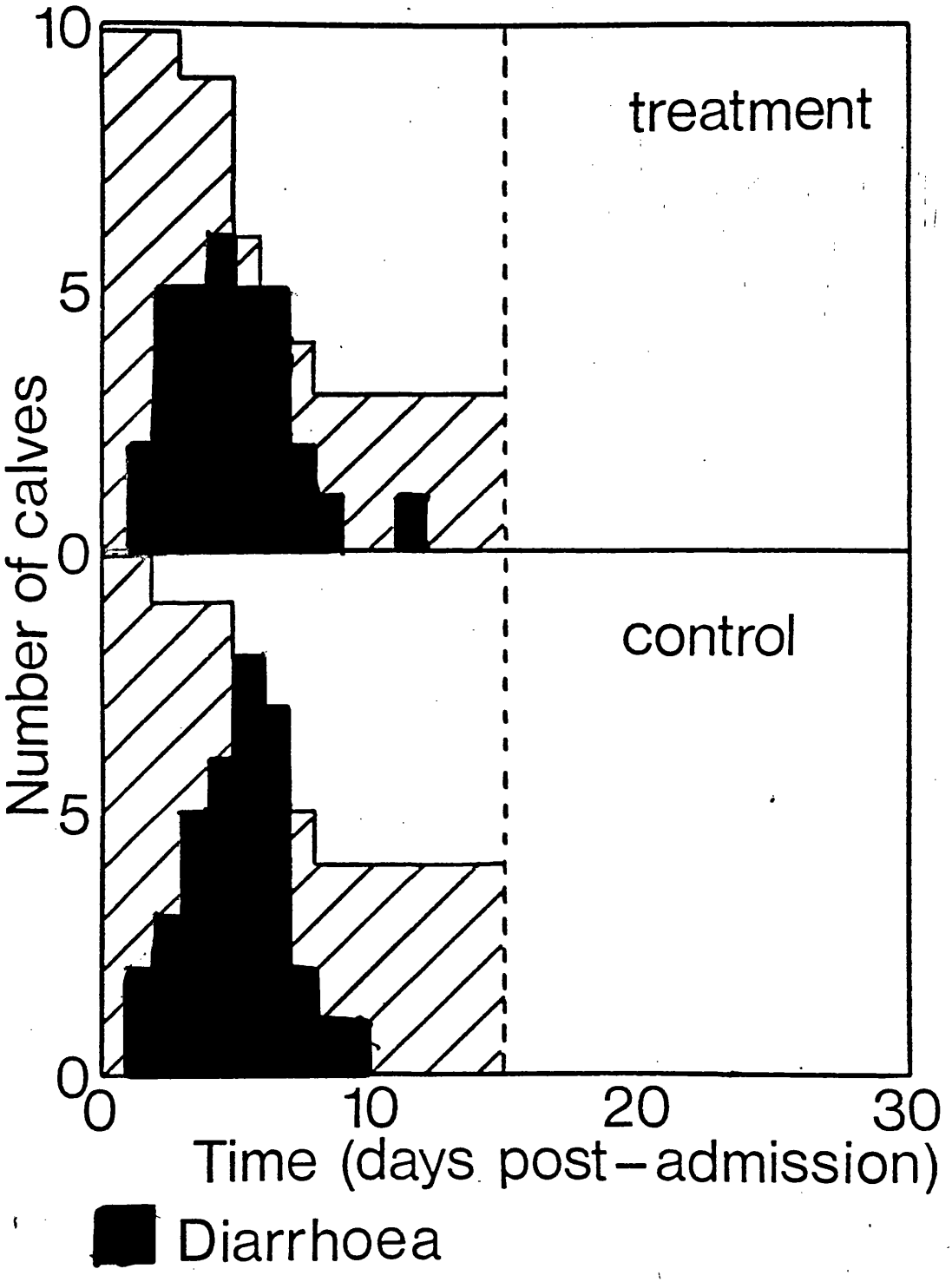
Morbidity

- Diarrhoea:

All calves in both groups developed severe diarrhoea at some time during the first week of the experiment and the peak prevalence was on day four (treatment calves) and day five (controls). There was very little difference in the extent of diarrhoea between the two groups but when calf/diarrhoea days were calculated as a percentage of the total number of live calves at any one time, the results were 35% (treatment calves) and 39% (controls) (see figure 10). As in previous experiments, there was widespread dullness among the calves of both groups during the first week but a general improvement occurred in most of the survivors towards the end of the second week.

No relationship was found between the degree or duration of diarrhoea and ZST values on admission. Moreover, an attempt (based on post-mortem findings) to differentiate between the five calves which appeared to have died as the result of uncomplicated diarrhoea (mean ZST, 9 units) and the eight which died either from diarrhoea and pneumonia or pneumonia *per se* (mean ZST, 8 units) yielded no useful information except insofar as it emphasised the very low ZST values in each of the groups (maximum treatment 22, maximum control, 24).

FIGURE 10 : THE PREVALENCE AND PATTERN OF DIARRHOEA AND MORTALITY IN THE TWO GROUPS OF TEN CALVES STUDIED IN EXPERIMENT 3


10
5
0
treatment
control
5
0
0
10
20
30
Number of calves
Time (days post–admission)
Diarrhoea

- Respiratory signs:

Three calves in the treatment group (44, 47, 49) and five calves in the control group (51, 53, 58, 59, 60) developed severe respiratory signs, all within 1-5 days after the onset of the trial.

- Umbilical infections:

Six calves in the treatment group (41, 43, 44, 46, 47, 50) and four controls (51, 53, 58, 60) developed umbilical abscesses. Although an attempt was made to treat this disorder as previously with parenteral procaine penicillin and streptomycin, all affected calves died from either diarrhoea or else diarrhoea and pneumonia before any response could be detected.

- Meningitis:

Two cases of meningitis occurred. Both of these calves had very low ZST values (3, 8 units) and it is possible that the meningitis arose following colisepticaemia; however, this was not confirmed on post-mortem examination.

Mortality

A total of 13 calves (65%) died in this experiment, all between days two and eight (see figure 10). Seven deaths (70%) were in the treatment group and six (60%) among the controls. The situation regarding possible relationships between diarrhoea, death and ZST values has already been discussed. Two calves had slightly blood-tinged faeces just prior to death.

Growth rates

The individual and mean growth rates for the calves in this trial are presented in Appendix 6. The mean weekly weight changes

FIGURE 11 : THE MEAN WEEKLY BODYWEIGHT CHANGES (EXPRESSED AS A PERCENTAGE OF MEAN ADMISSION WEIGHTS) OF THE TWO GROUPS OF TEN CALVES STUDIED IN EXPERIMENT 3

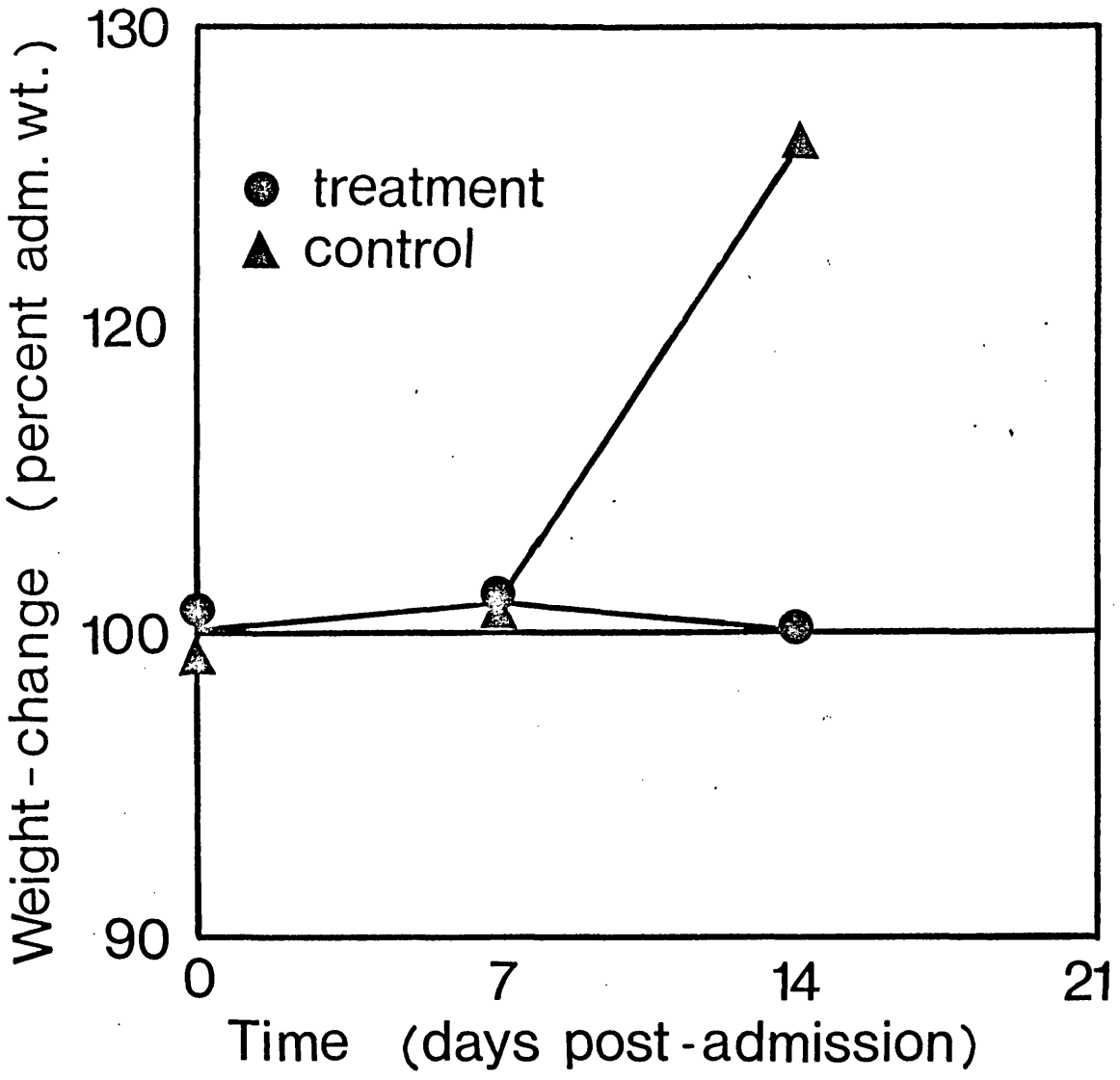

130
120
100
90
Weight-change (percent adm. wt.)
treatment
control
0
7
14
21
Time (days post-admission)

FIGURE 12 : WEEKLY WEIGHT CHANGES OF THE 20 INDIVIDUAL CALVES STUDIED IN EXPERIMENT 3

of both groups of calves are presented in figure 11 and it can be seen that the mean values are markedly (but not significantly) greater on days seven and fourteen than those of the treatment calves. However, these results should be viewed against a background of erratic weight changes (see figure 12) with one calf in each group growing relatively well at a time when most other calves were losing weight. The mean weight gains at 14 days post-admission were similar, 0.7 ± 3.1 kg (treatment calves) and 0.8 ± 4.4 kg (controls).

DISCUSSION

The third experiment in this series was similar to the second in that the calves were introduced into cleaned-out but not disinfected accommodation immediately after the departure of the previous group; similarly, the trial lasted only 14 days. The only significant difference in the experimental procedure was a change of dosage rate. In this third trial the calves received 150 mgm LBC twice daily for the first week and 75 mgm LBC twice daily for the second.

That the pathogenic challenge of the environment into which the experimental calves were introduced was gradually increasing seems likely in view of the steadily increasing overall diarrhoea rates from 22% to 40% to 41% and the increasing mortality rates, from 40% to 55% to 65% in experiments 1, 2 and 3 respectively. However, note should also be made of the fact that in the present experiment, the overall mean ZST value of 9 units was considerably lower than the corresponding figure (approximately 17 units) in experiments 1 and 2. This situation was unavoidable as a result of fewer calves being marketed at the time (April) when this experiment was commenced and a general tendency to low ZST in the calves which were initially acquired for further selection.

Diarrhoea affected all calves in this trial and the calf/diarrhoea rates in the two groups of calves were approximately equal (i.e. 39%, controls; 40% treatment calves). The mortality rate in the two groups were 70% (treatment group) and 60% (controls). The majority of calves which died in the treatment group, did so from the effects of diarrhoea although there was an overall greater number of deaths from the combined effects of diarrhoea and pneumonia than in the previous trials. No clearcut relationship could be found between ZST values and morbidity and mortality but, again, attention should be paid to the overall tendency for low ZST values in the calves in this trial and the fact that only two calves had values in excess of 20 units.

The mean weekly bodyweight of the control calves was greater (but not significantly so) than that of the calves which were treated. The major problem in analysing the bodyweight data was the rather erratic performances of certain individual calves in each group; on days seven and fourteen there were six and four calves respectively which had lost weight since admission. By the end of the trial the weight changes in the control calves ranged from (-)5 to (+)7 kg and in the treatment calves (-)2 to (+)5 kg. This situation also adversely affected any chance of gaining useful results from a statistical analysis of weight changes over the 14-day experimental period and the values were almost identical (i.e. control calves, 0.8 ± 4.4 kg; treatment calves, 0.7 ± 3.1 kg).

In short, the results of this experiment yielded no support for the view that LBC was useful in preventing or minimising neonatal calf diarrhoea. No significant difference could be demonstrated between groups in terms of the prevalence of diarrhoea nor growth

performances and the mortality rate in treated calves was higher than that in the controls. However, the effect of increasing pathogenic challenge as first demonstrated by Aschaffenburg and others (1949), particularly in the face of increased susceptibility must be looked upon as playing a major role in diminishing the efficacy of LBC which, after all, had been confirmed as being of significant value in the previous two studies.

SECTION 4: EXPERIMENT 4: AN INVESTIGATION INTO THE EFFECT OF FEEDING 150 mgm LBC ADMINISTERED IN RECONSTITUTED MILK SUBSTITUTE TWICE DAILY FOR SEVEN DAYS FOLLOWED BY 75 mgm IN RECONSTITUTED MILK SUBSTITUTE TWICE DAILY FOR A FURTHER SEVEN DAYS.

In the following experiment it was decided to investigate the effect of LBC dosed at the same rate as in experiment 3 to calves fed reconstituted milk substitute but otherwise selected and managed as in the previous experiments.

MATERIALS AND METHODS

The sources, types, method of selection and standardisation of the two groups of calves were as described previously.

On this occasion, 22 calves were acquired and admitted to the Department of Monday and Tuesday, May 7 and 8, 1979. Two of these were rejected since they were already ill on arrival. After selection, the mean body weight of the 20 experimental calves was 34.7 kg and the mean ZST value, 16.6 units. Repeated examination of faecal swabs failed to reveal the presence of Salmonellae.

Accommodation and grouping were similar to previous experiments and the arrangements are set out in figure 13. The trial commenced on Wednesday May 9, and lasted 28 days. Approximately one week prior to the admission of these calves the animals surviving from experiment 3 were removed and the pens were cleaned out, steam-jetted and disinfected.

All calves were fed on reconstituted milk substitute (BOCM 176, Gold top baby calf feed). This was mixed at the rate of 100 g/litre of water and this was fed warm at the approximate rate of 5% bodyweight, twice daily. The milk powder in question contained 10% oil, 23%

FIGURE 13

ACCOMMODATION AND GROUPING OF CALVES

Experiment 4

High Treatment (3) Calves Mean body-weight on admission 36.6 Kg. (Range = 32-40 Kg) Mean ZST Value on admission 30 units (Range = 23-39 units)		Control Low (3) Calves Mean body-weight on admission 30.3 Kg. (Range = 29-32 Kg) Mean ZST Value on admission 2.6 units (Range = 2-4 units)
Intermediate Treatment (4) Calves Mean body-weight on admission 34.5 Kg. (Range = 33-36 Kg) Mean ZST Value on admission 14.5 units (Range = 12-18 units)	CENTRAL FEEDING PASSAGE	Control Intermediate (4) Calves Mean body-weight on admission 38 Kg. (Range = 31-47 Kg) Mean ZST Value on admission 16.5 units (Range = 12-23 units)
Low Treatment (3) Calves Mean body-weight on admission 33 Kg. (Range = 30-37 Kg) Mean ZST Value on admission 5.6 units (Range = 1-9 units)		Control High (3) Calves Mean body-weight on admission 35 Kg. (Range = 35- Kg) Mean ZST Value on admission 30.6 units (Range = 23-41 units)

DOOR

protein and 0.5% fibre, according to information supplied by the manufacturers.

Treatment calves received 150 mgm LBC in their milk, twice daily for the first week of the experiment and 75 mgm twice daily for the second week.

Morbidity

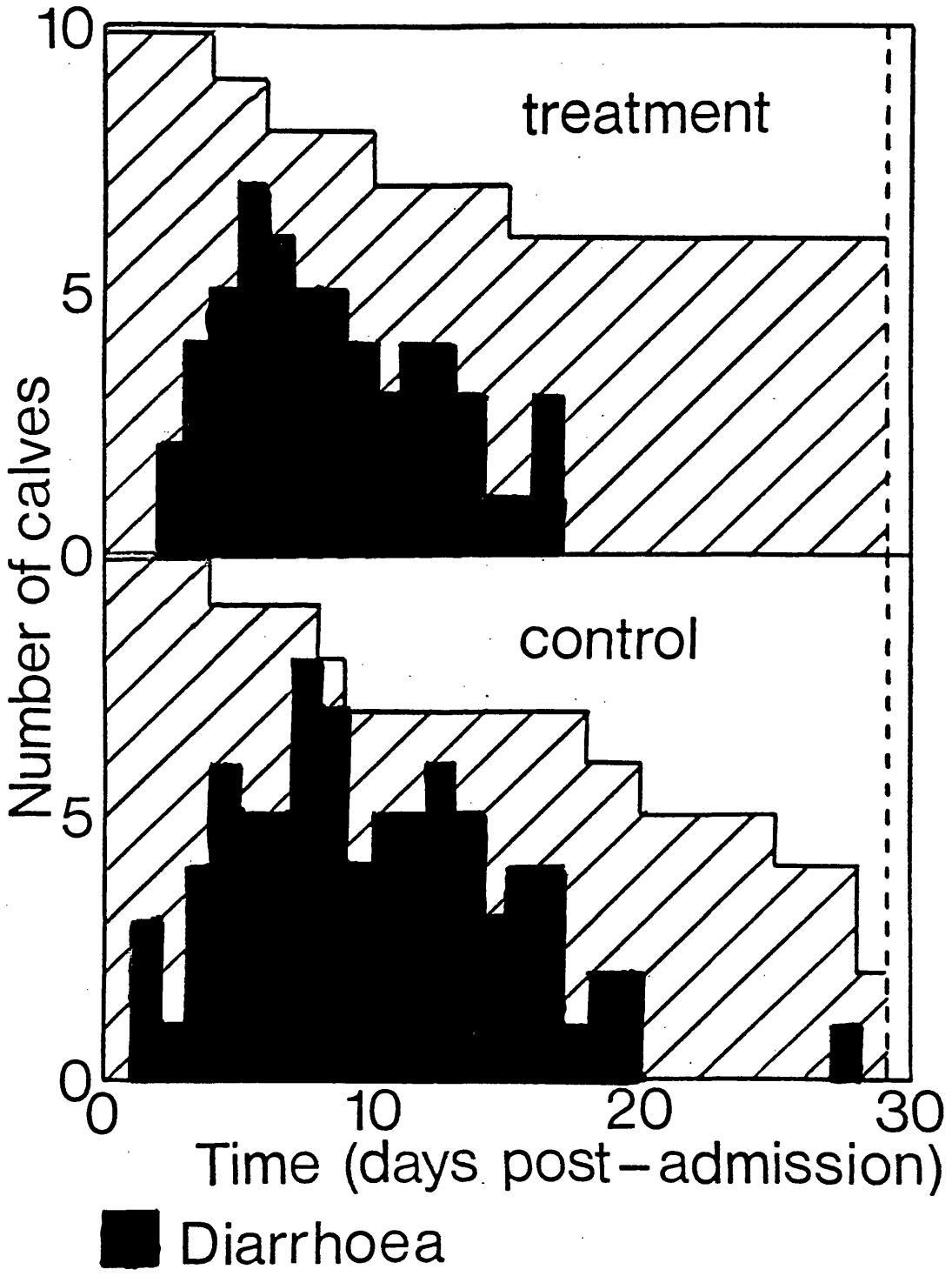
- Diarrhoea:

All calves in both groups developed severe diarrhoea sometime within the first week of the trial. The peak prevalence was on day four (treatment calves) and day six (controls). It can be seen from figure 14 that diarrhoea became much more prevalent in the control calves than in the calves receiving LBC and that this feature was most marked in the "low" ZST calves.

The prevalence of diarrhoea, as judged by the precentage of calf/diarrhoea days out of the total number of live calf days was found to be 28% (treatment calves) and 41% (controls). However, it should be remembered that in this instance, the experimental period lasted 28 days and while several deaths occurred during the last week, there was very little diarrhoea (see figure 14). Thus, for a more meaningful comparison with experiment 1, for example, it is useful to consider these values for the first 21 days of the trial; these were found to be 35% (treatment calves) and 49% (controls). Diarrhoea was more prolonged in the surviving control calves.

As the trial progressed, it became apparent that the survivors in the treatment group were much brighter and more alert than the controls.

FIGURE 14 : THE PREVALENCE AND PATTERN OF DIARRHOEA AND MORTALITY IN THE TWO GROUPS OF TEN CALVES STUDIED IN EXPERIMENT 4


10
5
0
treatment
control
5
0
Number of calves
0
10
20
30
Time (days post–admission)
Diarrhoea

Apart from the much higher prevalence of diarrhoea in the low ZST control calves (see figure 14), little more can be said regarding the relationship between ZST values and diarrhoea. Only two calves (70, 75) died of what appeared to be uncomplicated diarrhoea and their ZST values were 1 and 18 units, respectively.

- Respiratory signs:

Respiratory signs arose in ten calves between days one and twelve. In all of these calves, the signs gradually worsened until the time of death and pneumonic areas were found in the lungs of each calf.

- Conjunctivitis:

One calf (76) developed bilateral conjunctivitis which lasted four days.

Mortality

Twelve calves (60%) died during the experimental period of 28 days and the mortality pattern is summarised in figure 14 from which it can be seen that the corresponding figure over 21 days (i.e. as in experiment 1) would have been nine deaths (45%). In all, four treatment calves (40%) died compared with eight (80%) controls. There were distinct differences between the timing and cause of these deaths. The ZST values of the four treatment calves which died were 1, 9, 12 and 13 units (mean 9) and the animals died between days four and 15; one calf (70) was considered to have died from diarrhoea, one (66) from abomasal tympany and pneumonia and two (67, 68) from diarrhoea and pneumonia. In comparison, the ZST values of the eight controls which died were 2, 2, 4, 12, 13, 18, 23 and 28 units (mean 13); one (75) was thought to have died from diarrhoea alone, one (77) from pneumonia

alone and the rest from a combination of the two problems. In these eight control calves, the mortalities were spread over a period from day four to day 28 and five (all pneumonic or pneumonic and diarrhoeic) died after day 15 (see figure 14).

- Growth rates:

Individual and mean weekly weights for the calves in this experiment are presented in Appendix 8. The mean weekly weight changes are presented in figure 15 from which it can be seen that marked differences were present from day 14 until the end of the experimental period with the treated calves weighing considerably more than the controls on every occasion. The mean weekly weights on day 14; treatment calves, 35.0 $\pm$ 2.8 kg, controls, 32.0 $\pm$ 4.8 kg; day 21: treatment calves, 39.5 $\pm$ 3.2 kg, controls, 33.8 $\pm$ 5.9; day 28: treatment calves, 44.2 $\pm$ 3.3 kg, controls 42.0 $\pm$ 1.0 kg. However, statistical analysis revealed that none of these differences were significant.

The individual weekly weight changes during the trial are summarised in figure 16. It can be seen that a substantial number of calves lost weight over the first three weeks of the trial and the majority of these were always controls. From day 21 onwards, marked weight gains occurred in seven calves only one of which was a control animal. An analysis of the weight changes revealed that the values on day 14 were: treatment calves, (-) 0.7 $\pm$ 0.94 kg, controls (-) 2.9 $\pm$ 2.59 kg and the day 21 changes were: treatment calves, 3.8 $\pm$ 0.9 kg, control calves, (-) 1.0 $\pm$ 1.67 kg. The day 14 values were not significantly different ($p = 0.08$) but the day 21 differences were highly significant ($p = 0.002$).

FIGURE 15 : THE MEAN WEEKLY BODYWEIGHT CHANGES (EXPRESSED AS A PERCENTAGE OF MEAN ADMISSION WEIGHTS) OF THE TWO GROUPS OF TEN CALVES STUDIED IN EXPERIMENT 4

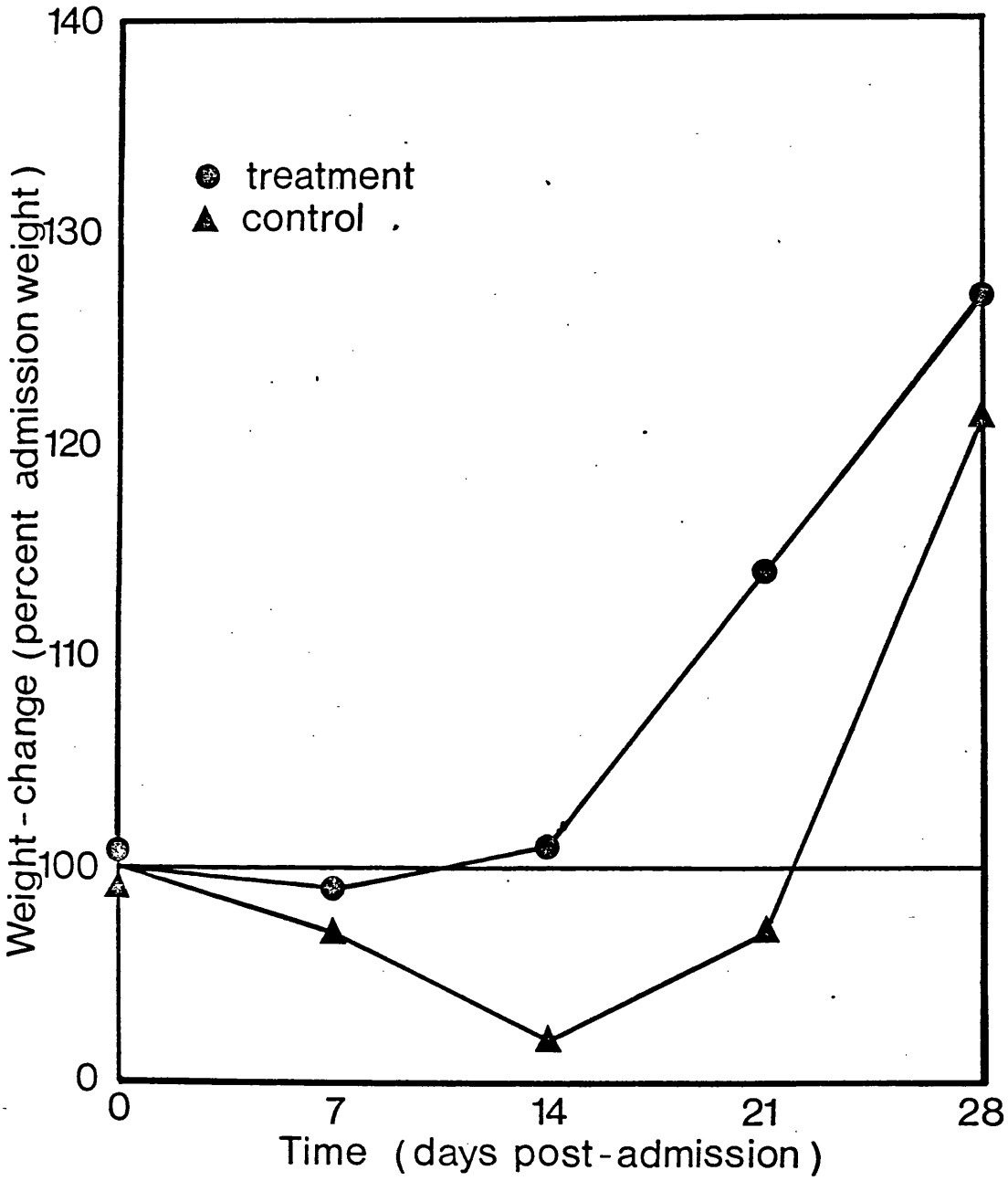
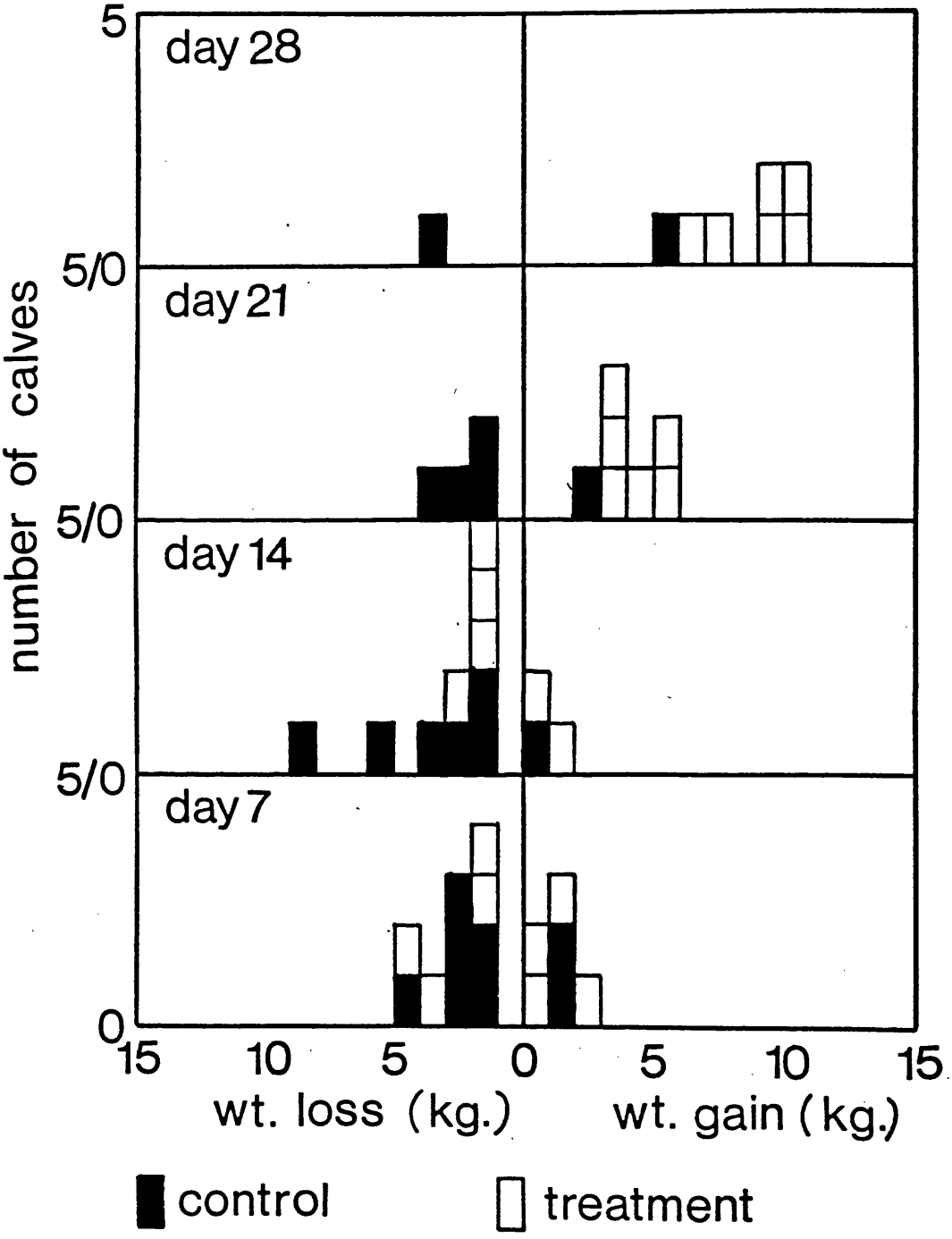

140
130
120
110
100
0
Weight-change (percent admission weight)
treatment
control
0
7
14
21
28
Time (days post-admission)

FIGURE 16 : WEEKLY WEIGHT CHANGES OF THE 20 INDIVIDUAL CALVES STUDIED IN EXPERIMENT 4


5
day 28
5/0
day 21
5/0
day 14
5/0
day 7
0
number of calves
15
10
5
0
5
10
15
wt. loss (kg.)
wt. gain (kg.)
control
treatment

DISCUSSION

This experiment was designed to investigate the value of LBC in preventing, or minimising, neonatal diarrhoea in calves fed reconstituted milk substitute and the dose rate chosen was 150 mgm twice daily for the first week and 75 mgm twice daily for the second week of the trial after which time supplementation ceased. Apart from the change of diet and the fact that the study was carried out over 28 days, the conditions were the same as in Experiments 1, 2 and 3. However, in view of the extremely high morbidity and mortality rates in the previous study and the consequent difficulties involved in assessing the value or otherwise of LBC, the calf accommodation was cleaned out after the departure of the previous group of calves, steam-jetted and then disinfected. The room was then rested, but only for six days, prior to the arrival of the 20 calves used in this study. The overall mean ZST value was 17 units and hence the same as that in Experiments 1 and 2.

Diarrhoea affected all individuals in both groups within one week of the start of the study. However, the percentage of calf/diarrhoea days was found to be much higher in the controls (41%) than in the treatment groups (28%). If the 21-day figures are examined in order to carry out a more valid comparison with the data obtained in Experiment 1, the corresponding figures are 49% and 35% respectively; thus diarrhoea was more prevalent in these milk powder-fed calves than in the calves fed on raw cow's milk. Diarrhoea was far more prevalent in the untreated calves with low ZST values than in their treated equivalents. The mortality rate was much higher in the control calves (80%) than in those which received LBC (40%). and, there was a clearcut relationship between death and ZST values. The mean value of fatal (treated) cases was found to be 9 units while that of survivors was

22 units; the results in the control calves were: fatal cases, 13 units, survivors, 32 units, although, again, five of the deaths in the control calves occurred between days 18 and 28, much later than in the treatment groups or in both groups in Experiment 1.

The mean weekly bodyweight changes revealed distinct differences between groups from day seven onwards, although none of these differences were found to be statistically significant. Weight losses were prominent in both groups on days 7 and 14 but was restricted to control calves on days 21 and 28. Again, the lack of statistical significance may be attributed, at least in part, to the erratic growth performances in both groups of calves and to the steadily diminishing size of the control group due to mortality. There seems little doubt that the weight changes reflect the severity and/or duration of diarrhoea (or diarrhoea plus respiratory disease), particularly in view of the fact that as the prevalence of diarrhoea decreased and some diarrhoeic calves died, so mean weight gains markedly increased.

That the initial weight losses or poor growth rates were due to diarrhoea is further supported by the steady weight gains in the surviving treated calves between days 14 and 28. Despite the fact that all of them had been affected by some degree of diarrhoea their mean weekly weight increases over this period was 4.6 kg/calf/week, thus indicating that the milk powder in question and its method of preparation and administration were quite adequate. The mean weight changes up to day 14 were actual losses in both groups but the differences were not significant ($p = 0.08$) whereas most of the treatment calves had gained weight by day 21 and the difference between groups at this stage was very highly significant ($p = 0.002$).

In summary, severe diarrhoea affected both groups of calves and was responsible for many calves losing weight during the first half of the 28 day experimental period. However, it was more prevalent, and prolonged, in the untreated calves and depression of weight gain and/or weight loss were more profound. The mortality rate was much higher in the untreated control calves and many deaths occurred relatively late in the course of the study. The mean weight gains to 21 days were higher in treated calves and once diarrhoea was past the surviving-treated calves grew at an acceptable rate despite their earlier setbacks.

SECTION 5: EXPERIMENT 5: AN INVESTIGATION INTO THE EFFECTS OF FEEDING LBC AT THE RATE USED IN EXPERIMENT 4 TO CALVES FED EITHER RAW MILK OR POWDERED MILK SUBSTITUTE.

The final experiment in this programme was designed to study the effect of feeding LBC at the rates used in Experiments 3 and 4 to calves fed either raw cow's milk or reconstituted milk powder but otherwise managed under standardised conditions.

MATERIALS AND METHODS

The procedures were as in previous experiments except that an effort was made to acquire as many calves as possible with high ZST values. Thus, a total of 33 calves were obtained in local markets and all but 16 were rejected mainly because of relatively low ZST values. After selection the mean bodyweights of the 16 experimental calves was 34.1 kg and the mean ZST value 29.8 units. Repeated examination of faecal swabs revealed the presence of Salmonella worthington in one calf (89) on one occasion only.

The housing arrangements were as reported previously and are summarised in figure 17. The room which contained the calves had been cleaned, steam-jetted and disinfected prior to the arrival of the experimental animals. The trial commenced on Wednesday, July 25, 1979.

One group of eight calves were fed raw cow's milk at the same rate as in Experiment 1. The other group were fed reconstituted milk substitute of the same type and at the same rate as in Experiment 4. The dose rate of LBC was as in Experiment 4 and, again, the powder was administered in the milk.

FIGURE 17

ACCOMMODATION AND GROUPING OF CALVES

Experiment 5

High Powder Milk (3) Calves Mean body-weight on admission 34 Kg. (Range = 32-36 Kg) Mean ZST Value on admission 36.3 units (Range = 33-40 units)		Raw Milk Low (3) Calves Mean body-weight on admission 32 Kg. (Range = 23-40 Kg) Mean ZST Value on admission 23.3 units (Range = 21-26 units)
Intermediate Treatment (2) Calves Mean body-weight on admission 33.5 Kg. (Range = 30-37 Kg) Mean ZST Value on admission 28 units (Range = 28-28 units)	CENTRAL FEEDING PASSAGE	Control Intermediate (2) Calves Mean body-weight on admission 38 Kg. (Range 2 36-40 Kg) Mean ZST Value on admission 29 units (Range = 28-30 units)
Low Treatment (3) Calves Mean body-weight on admission 34.3 Kg. (Range = 27-41 Kg) Mean ZST Value on admission 24.6 units (Range = 21-27 units)		Control High (3) Calves Mean body-weight on admission 34 Kg. (Range = 33-35 Kg) Mean ZST Value on admission 37 units (Range = 35-38 units)

DOOR

RESULTS

Morbidity

- Diarrhoea:

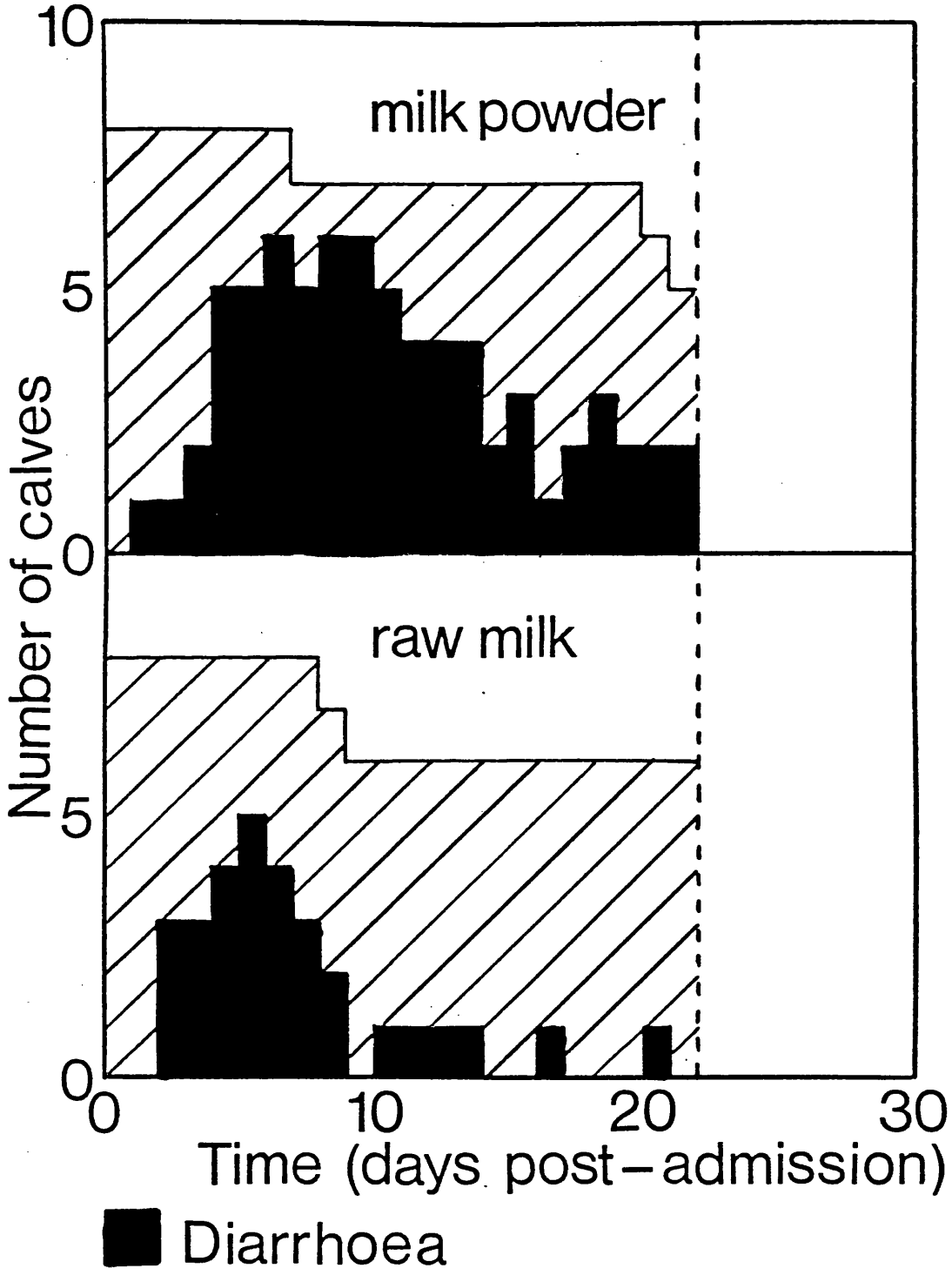
All calves save one (89) developed diarrhoea at some time during the experiment although, as can be seen in figure 18, the prevalence of diarrhoea was much higher in the calves fed milk substitute. The calves with low ZST values and fed milk substitute seemed particularly prone to developing diarrhoea. The peak prevalence of diarrhoea in the milk-fed calves was day 4 and in the substitute-fed calves was between days 5 and 8.

The number of calf/diarrhoea days in the milk fed calves was 30, that is 20% of 149 possible live-calf days; the corresponding figures for the substitute-fed calves was 69 of 158 days (44%). The major difference was partly due to the fact that diarrhoea was much more prevalent in the substitute-fed calves during the early part of the trial and partly because the problem persisted in several calves up until day 20.

Increasingly as the trial progressed the calves fed cow's milk appeared to be brighter, cleaner and in better health, mainly because progressively fewer were diarrhoeic.

Two calves (88, 92) were considered to have died as a result of diarrhoea and the remainder (85, 87, 94) succumbed to a combination of diarrhoea and pneumonia. Diarrhoea tended to be of shorter duration (range 3-6 days) in the milk-fed fatal cases than in the substitute-fed fatal cases (range 4-13 days).

FIGURE 18 : THE PREVALENCE AND PATTERN OF DIARRHOEA AND MORTALITY IN THE TWO GROUPS OF EIGHT CALVES STUDIED IN EXPERIMENT 5


10
milk powder
5
0
raw milk
Number of calves
5
0
0
10
20
30
Time (days post–admission)
Diarrhoea

- Respiratory signs:

Respiratory disease was not as widespread in the calves in this experiment compared with earlier groups. One calf (94) was noted to be coughing on day 3 and two others (85, 87) on day 12; and three calves eventually died, from the effect of severe, prolonged diarrhoea and anterior lobe pneumonia.

Mortality

In all, five calves (31%) died; two which were in the milk-fed group (25%) and three in the substitute-fed group (38%).

The deaths in the milk-fed calves were relatively early, on days 8 and 9. One calf (92) was thought to have died from diarrhoea, the other (94) from a combination of diarrhoea and pneumonia. One calf in the substitute-fed group (88) died on day 7 apparently from diarrhoea, whereas the other two (85, 87) died of a combination of diarrhoea and pneumonia on days 20 and 21.

In this group of calves selected for high ZST values, there was no obvious relationship between the mortality pattern and ZST units.

Growth rates

The individual and mean weekly weights of the calves in this experiment are presented in Appendix 10. It can be seen from figures 19 and 20 which summarise this information, that distinct differences existed between the mean weekly weight changes of the two groups of calves from day 7 onwards, with the milk-fed calves always gaining weight fastest. The mean values in question were as follows: day 7 - milk-fed calves, 34.3 ± 4.7 kg; substitute-fed calves, 33.3 ± 4.7 kg; day 14 - milk-fed calves, 36.7 ± 5.3; substitute-fed calves, 31.3 ± 5.5 kg; day 21 - milk-fed calves, 41.5 ± 5.8 kg;

FIGURE 19 : THE MEAN WEEKLY BODYWEIGHT CHANGES (EXPRESSED AS A PERCENTAGE OF MEAN ADMISSION WEIGHTS) OF THE TWO GROUPS OF EIGHT CALVES STUDIED IN EXPERIMENT 5

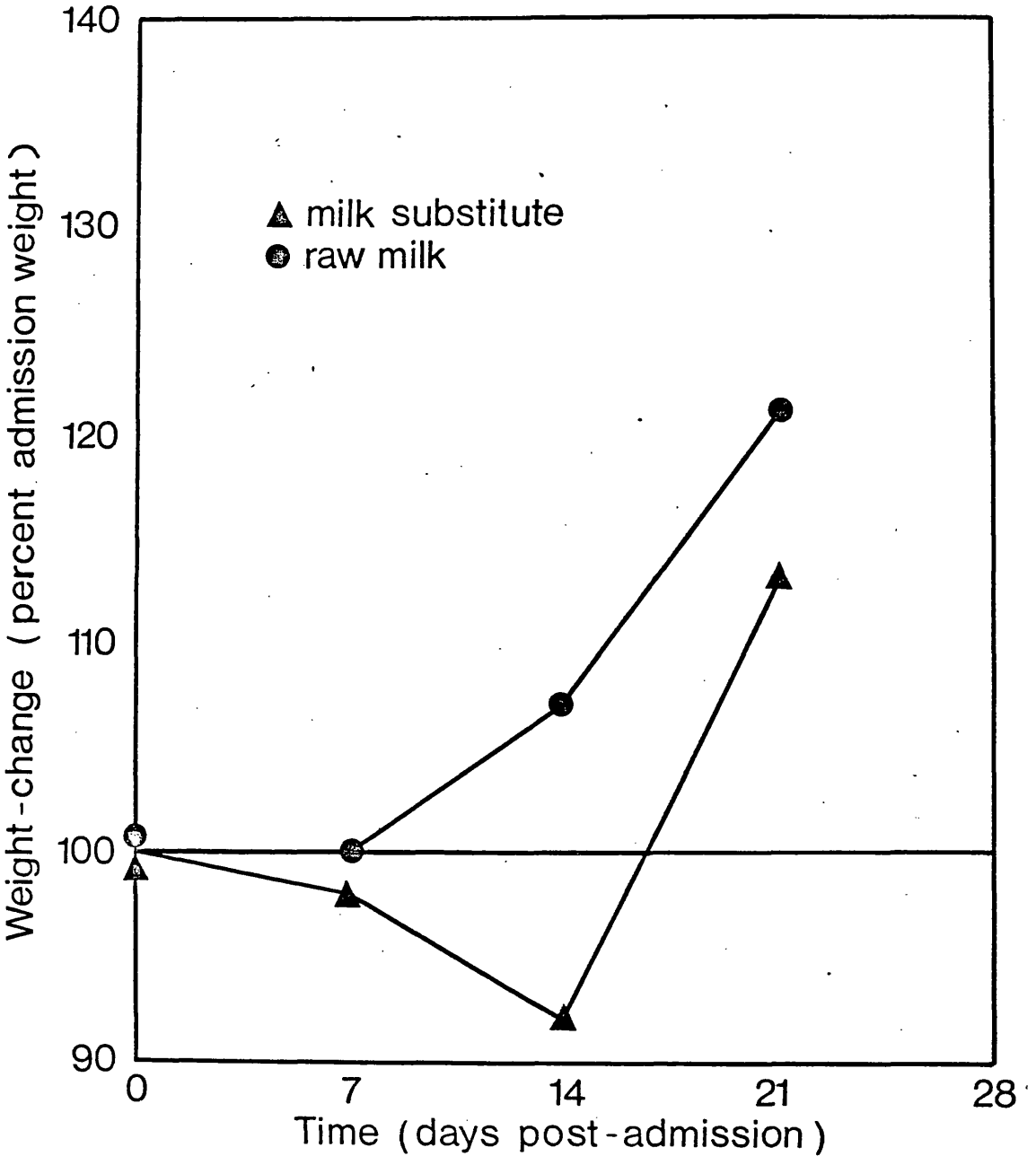
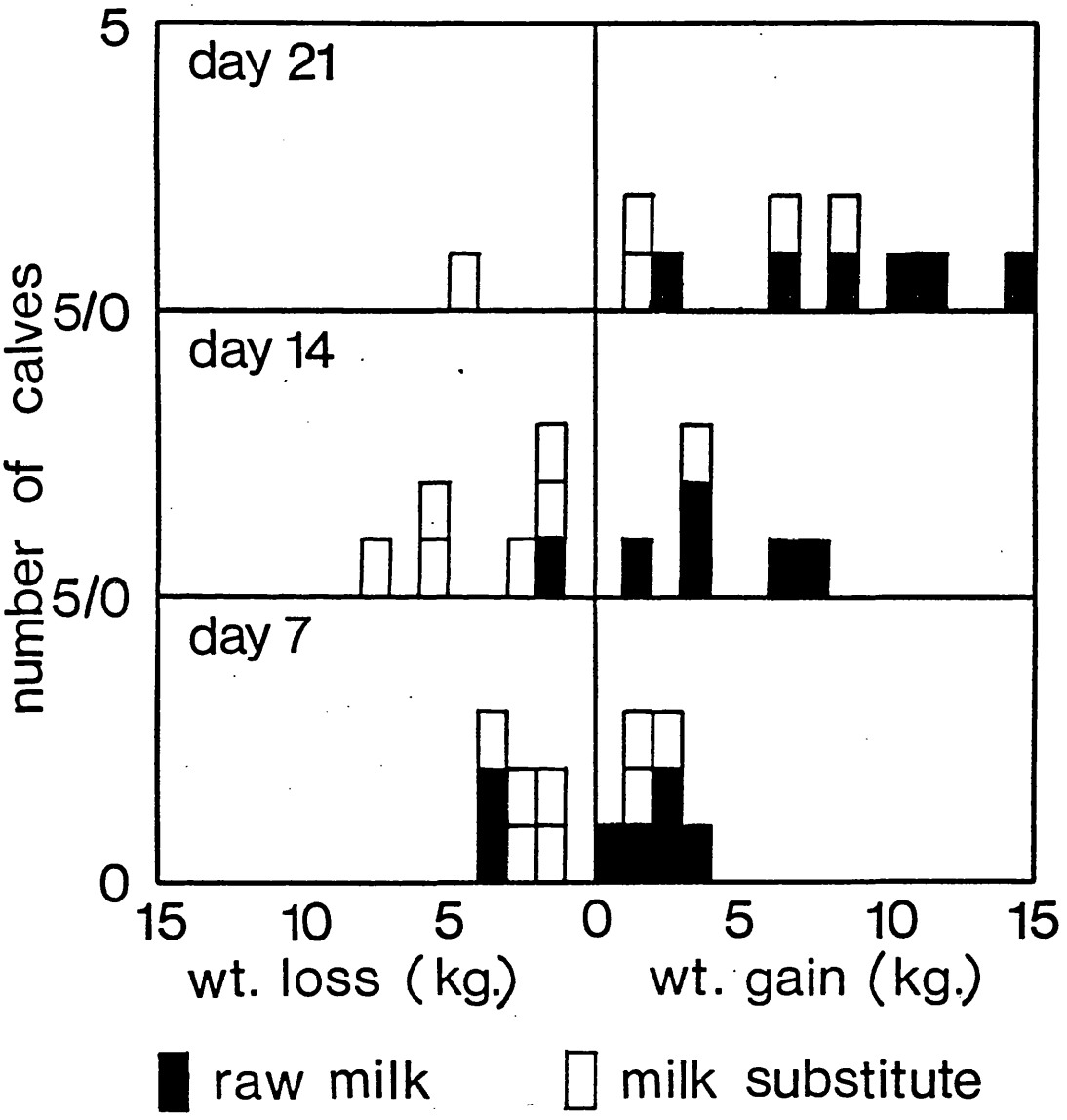

140
130
120
110
100
90
Weight-change (percent admission weight)
milk substitute
raw milk
0
7
14
21
28
Time (days post-admission)

FIGURE 20 : WEEKLY WEIGHT CHANGES OF THE 16 INDIVIDUAL CALVES STUDIED IN EXPERIMENT 5


5
day 21
5/0
day 14
5/0
day 7
0
number of calves
15
10
5
0
5
10
15
wt. loss (kg.)
wt. gain (kg.)
raw milk
milk substitute

substitute-fed calves, 38.4 $\pm$ 5.8 kg. However, none of these values were found to be significantly different.

The findings regarding individual weight changes over the experimental period are summarised in figure 20. It can be seen that on days 7 and 14, a substantial number of calves weighed less than their admission weights and on both occasions the majority of these were substitute-fed calves. By day 21, the surviving calves were mostly gaining weight but the calves being fed raw milk were still ahead of the substitute-fed animals. Investigations revealed that the mean weekly weight changes on day 14 were: milk-fed calves, 3.7 $\pm$ 3.5 kg; substitute-fed calves, (-) 2.6 $\pm$ 3.1 kg, and on day 21 were: milk-fed calves 8.5 $\pm$ 3.8 kg; substitute-fed calves, 2.4 $\pm$ 4.2 kg. Both of these differences were found to be statistically significant ($p = 0.01$ and $p = 0.05$, respectively).

DISCUSSION

This fifth and final investigation was designed with the aim of examining what, from the results obtained in Experiments 1 and 4, seemed to be a distinct difference in the prevalence of diarrhoea depending on whether raw milk or reconstituted milk powder was fed.

The calf accommodation was cleared of calves soon after the termination of Experiment 4 and it was then cleaned out, steam-jetted, disinfected and "rested" for approximately one month. It was decided to use as many calves as possible with high ZST values and this, together with the relative dearth of suitable marketed calves in July, was responsible for the fact that the group sizes were limited to eight. The experimental period was 21 days and the dosage rate of LBC was maintained at the rate used in the previous experiment. The preparation was fed to both groups of experimental calves.

It soon became evident that the prevalence of diarrhoea was much higher in the calves fed milk powder and by the end of the trial the powder-fed calves were found to have a calf/diarrhoea percentage of 44% compared with a figure of 20% in those fed raw cow's milk. In addition, diarrhoea was present in a substantial number of milk powder-fed calves throughout the 21 day experimental period whereas it followed the more established pattern of peaking at around the fourth day in the milk-fed calves. The mortality rate was found to be 25% in these latter animals compared with 38% in the group fed reconstituted milk powder. Again, two of the three deaths in the milk powder-fed group occurred towards the end of the experimental period.

After the first week, when the mean bodyweight of the milk-fed calves remained static, there was a steady increase and at all times the mean weekly values were well in excess of the calves fed milk powder. Indeed, these latter animals suffered steadily decreasing mean values until day 21 when it was found that the five survivors were at last gaining weight. Despite the marked differences in performances of the two groups, highlighted as it was by the fact that on day 7, five of the seven, and on day 14, six of the seven calves to have lost weight were powder-fed, it was not possible to demonstrate a statistically significant difference between the values obtained. However, an examination of the weight changes between the start of the experiment and days 14 and 21 revealed highly significant differences ($p = 0.01$, $p = 0.05$ respectively) and, again, these differences can be ascribed, at least in part, to the differing prevalence of diarrhoea in the two groups.

In summary, despite the differences highlighted between the test and control (milk powder-fed) calves in the previous experiment, clear differences were demonstrated in the fifth trial between two groups of calves (both of which were administered LBC) depending on whether they were fed reconstituted milk powder or raw cow's milk. Fewer of the calves fed raw cow's milk died, they also suffered less diarrhoea, and their mean weight gains by days 14 and 21 of the trial were significantly greater than the powder-fed animals.

CHAPTER 4

GENERAL DISCUSSION

GENERAL DISCUSSION

A study of the relevant literature confirms that neonatal calf diarrhoea is widely acknowledged as being a major economic burden in cattle-rearing operations in many countries. It is a particular problem under conditions of intensive livestock management but it is not exclusive to such enterprises and, more sporadically, it may cause considerable losses and inconvenience under more extensive situations. In most outbreaks, an aetiological diagnosis is not attempted, let alone achieved, although clinical and epidemiological data usually suggest that the majority of outbreaks involve a considerable number of common features (Blood and others, 1979) thus perhaps pointing to the possibility that most outbreaks are initiated by a limited number of pathogens acting either alone or in concert (Acres, 1977).

The treatment of individual, severely-affected cases of neonatal calf diarrhoea under field conditions is often unsuccessful irrespective of whether antibiotics and fluid and electrolyte therapy are used alone or in combination (Radostits, 1965). Thus, attention has been drawn repeatedly to the possibility of preventing the problem or at least minimising its effects.

The protective role of colostrum has received a great deal of attention in recent years and it is now widely considered to be the dominant factor involved in whether or not calves survive the neonatal period (Selman, 1973). Thus, advice to calf producers must always involve detailed information about the colostral immunoglobulin absorption process and also how to minimise pathogenic challenge. In many cases such an approach is sufficient to control a calf diarrhoea problem (Selman, 1973). However, when attempts are being made to

rear calves which have been purchased or otherwise acquired within the first week or two of life and where the chances of controlling the first few critical hours of a calf's life are limited or non-existent, alternative preventive regimes must be considered.

Little sound evidence exists to support the routine practice of continuously or strategically dosing calves with antibiotics and the approach involving the vaccination of either calves or their dams has generally yielded either disappointing results or else information which is extremely difficult to assess (Acres, 1977; Blood and others, 1979). The major drawbacks would appear to be (i) the lack of detailed knowledge regarding the relative prevalence of, and roles played by, suspected pathogens, (ii) the questionable immune competence of very young calves, (iii) the age incidence of the common neonatal disorders and (iv) (in dam vaccination studies) the fact that so often the major problem seems to be suboptimal colostral immunoglobulin absorption irrespective of whether or not specific and valid antibodies are contained in the colostrum being presented to and/or ingested by the calf.

Another factor which is causing increasing concern is the fact that widespread and often haphazard long-term administration of antibiotics to young calves may be responsible, if only in part, for the increase in antibiotic resistance (Martinez, 1974). Thus, the gradual accumulation of information regarding the use of other approaches, and particularly, that involving the oral administration of certain lactic acid bacteria represents a useful alternative method of prophylaxis apparently without the unpleasant and dangerous effects referred to above (Beslin, 1975).

The information regarding the use of various preparations and formulations of lactic acid bacteria, generally administered in either milk or milk powder, to young pigs and calves has been extensively reviewed. This information strongly suggests that the approach is useful although the data regarding its use in pigs is rather more convincing and, apparently, less controversial than the use of these preparations in calves. It is for this reason that one such product, a lyophilised preparation of Strep. faecalis was investigated under conditions which were designed to standardise many of the variables which have made so many earlier studies into calf diarrhoea prophylaxis virtually impossible to assess (Acres, 1977).

In five separate investigations, strenuous efforts were made to control the source, type and size of the experimental calves, their immunological (ZST) status, the conditions of feeding and management that they encountered (at least while they were on test) and the nature and level of pathogenic challenge. In this latter respect, no attempt was made to define the basic cause of the high morbidity and mortality which occurred although by careful consideration of housing arrangements, at least it was ensured that all calves must have received a similar challenge.

The results of Experiments 1, 2 and 4 confirmed that the administration of the preparation in question (LBC) to test calves resulted in fewer deaths during the experimental periods of between 14 and 28 days, less severe diarrhoea and greater weight gains than that experienced by similar but untreated control calves. Thus, the results were in line with those of certain other workers (Meszaros and Varga, 1975; Milic and Sljivovacki, 1975; Radulovic and others, 1976; Tournut and others, 1976) who have used various lactic bacteria but often under

far less stringent experimental conditions than used in this present series.

Certain other observations made on the untreated control calves and on the events which occurred in Experiments 2, 3 and 5 are also worthy of comment and may also be of major importance in ensuring that future studies on the efficacy of LBC and similar preparations are carried out under optimal conditions.

Without doubt, the practice of not disinfecting between the groups of calves used in Experiments 1, 2 and 3, the fact that each study was run more-or-less concomitantly, and the presence right from the start of Experiment 1 of widespread diarrhoea led to a rapidly escalating pathogenic challenge that totally overwhelmed the calves in Experiment 3 and resulted in equivocal results in terms of the efficacy of LBC. With a return to normal cleaning, disinfection and resting practices, the situation reverted in Experiments 4 and 5 to something more akin to that experienced in Experiment 1. The fact that this increase in challenge happened to coincide with the selection of two groups of calves with lower mean ZST values than in the previous two experiments is also likely to have been of major importance in this respect. Such a situation has been postulated as being responsible for the seasonality of losses from neonatal calf disease in dairy herds in the west of Scotland (Selman 1969; 1973).

The other factor of major importance was that the prevalence of diarrhoea was closely related to the diet being fed. The prevalence of diarrhoea was greater in calves fed reconstituted milk powder and, in Experiment 5, calves fed in this way experienced more deaths, more diarrhoea and grew significantly slower than calves

fed raw cow's milk. Since this difference was apparent even when both groups were dosed with LBC, it seems unlikely that it was due to the presence of naturally-occurring lactic acid bacteria in the raw cow's milk in contrast to its total absence in the unsupplemented milk powders. The difference in prevalence of diarrhoea did not represent merely a difference in faecal consistency, since more powder-fed calves died and there was a far greater tendency to lose weight in the earlier part of the experiment. The milk substitute in question was a highly reputable product from a major farm feeds company and it seems unlikely that it was being produced by the inferior methods described by Roy (1969). Clearly, in view of the widespread use of milk powders in modern calf-rearing enterprises, more work should be carried out into this question, since it is just possible that it might be resolved by simply increasing the dose-rate of LBC.

Without doubt, the experimental model used in this series of investigations was an extremely harsh test system. It is tempting to believe that under better conditions of housing, husbandry and feeding and with calves somewhat less vulnerable to the adverse effects of neonatal infections than Ayrshire calves marketed when only a few days old, the results of LBC administration might well be much more impressive. For this reason it does seem that further work should be carried out in order to study the efficacy of the preparation under different management systems. Furthermore, in view of the evidence that diet and diarrhoea were strongly interrelated, attention should be paid to the value of LBC and similar products when supplementing various milk powder formulations. The current vogue for feeding "acid" milk substitutes (Low, 1978, 1979a,b) is of particular interest in this context since many of the suggested effects would appear to be similar in nature to those of the lactic acid bacteria.

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APPENDIX I : CERTIFICATE OF BACTERIOLOGICAL ANALYSIS FOR LBC CONCENTRATE M10 (LYOPHILISED STREPTOCOCCUS FAECIUM)

Microbiological Laboratory

Cert.-No.: 191

CERTIFICATE OF BACTERIOLOGICAL ANALYSIS

Preparation or goods	:	LB Concentrate M10
Internal No. of preparations or goods	:	839720
Batch-No. of Streptococcus faecium SF68	:	901/27
Microorganisms to determinate	A :	Total content of bacteria
	B :	Content of Coliforms
	C :	Content of Streptococcus faecium
Substrates used for determination	A :	Tryptone Glucose Extract Agar
	B :	Violet Red Bile Agar
	C :	Tomato Juice Agar
Results of analysis	A :	not less than $35 \cdot 10^9$/g
	B :	negative/0.1g
	C :	not less than $35 \cdot 10^9$/g
Time and temperature used for cultivation	:	48h/37°C
Dilution used for determination	:	$10^{-1}/10^{-9}$

Date of Certificate: Barbengo, 16.2.79

Signature of analyst

Checked by

APPENDIX 2 : SUMMARY OF DATA REGARDING SOURCE, ZST VALUES, WEIGHT CHANGES, GROUP ALLOCATION AND TREATMENT OF 20 INDIVIDUAL CALVES STUDIED IN EXPERIMENT 1.

EXPERIMENT 1

TREATMENT	150mgm LBC twice daily in feed; 21 days						CONTROL							
Calf No.	Market	ZST	Bwt. (Kg) 7-2	Bwt. (Kg) 14-2	Bwt. (Kg) 21-2	Bwt. (Kg) 28-2	Wt. Gain	Calf No.	Market	ZST	Bwt. (Kg) 7-2	Bwt. (Kg) 14-2	Bwt. (Kg) 21-2	Bwt. (Kg) 28-2	Wt. Gain
High								High							
1	Lanark	30	30	36	40	45	+15	11	Paisley	28	38	40	44	48	+10
2	Lanark	24	33	37	37	40	+ 7	12	Paisley	26	34	37	40	45	+11
3	Lanark	22	32	36	40	46	+14	13	Paisley	21	34	-	-	-	-
Mean		25.3	31.7	36.3	39.0	43.7		Mean		25.0	35.3	38.5	42.0	46.5	
Intermediate								Intermediate							
4	Lanark	20	36	40	45	48	+12	14	Lanark	20	32	-	-	-	-
5	Lanark	16	28	-	-	-	-	15	Lanark	18	32	-	-	-	-
6	Lanark	14	32	36	40	46	+14	16	Lanark	13	39	40	41	45	+ 6
7	Lanark	12	32	-	-	-	-	17	Lanark	12	29	-	-	-	-
Mean		15.5	32.0	38.0	42.5	47.0		Mean		15.7	33.0	40.0	41.0	45.0	
Low								Low							
8	Paisley	11	36	-	-	-	-	18	Paisley	12	36	40	41	48	+12
9	Lanark	8	35	36	40	40	+15	19	Lanark	8	34	-	-	-	-
10	Lanark	7	40	45	48	55	+15	20	Lanark	7	36	40	41	48	+12
Mean		8.7	37	40.5	44.0	47.5		Mean		9.0	35.2	40.0	41.0	48.0	
MEAN		16.4	33.4	38.0	41.4	45.7	11.7	MEAN		16.5	34.4	39.4	41.4	46.8	10.2

APPENDIX 3 (TABLES 1-20) : INDIVIDUAL DATA SHEETS FOR 20 CALVES USED IN EXPERIMENT 1, INCLUDING INFORMATION REGARDING PRESENCE (AND DEGREE) OR ABSENCE OF DIARRHOEA, SURVIVAL TIME AND GROSS POST MORTEM FINDINGS WHERE APPLICABLE.

Calf No. 1 Assigned group: Treatment Raw Milk

Admission Date: 7.12.1979

Body Wt. (Kg) on admission: 30

Zinc Sulphate Turbidity units: 30

LBC Dosage: 150mg.-feed-twice daily

Duration of administration: 21 days

Survival Time: 21 days

Observation Period: 21 days

Diarrhoea days:

0 0	1 0	2 0	3 0	4 +++	5 +++	6 +	7 0
8 0	9 0	10 0	11 0	12 +	13 0	14 0	15 0
16 0	17 0	18 0	19 0	20 0	21 0	22	23
24	25	26	27	28	29	30	31

Body weight changes: Kg.

0 30	7 35.9	14 39.5	21 45	28

Cause of death: -

Post-mortem findings: -

Comments: Lacrimation, umbilical swelling.

Calf No. 2 Assigned group: Treatment Raw Milk

Admission Date: 7.2.1979

Body Wt. (Kg) on admission: 32.7

Zinc Sulphate Turbidity units: 24

LBC Dosage: 150mg.-feed-twice daily

Duration of administration: 21 days

Survival Time: 21 days

Observation Period: 21 days

Diarrhoea days:

0 0	1 0	2 0	3 0	4 0	5 +	6 +++	7 +
8 ++	9 0	10 0	11 0	12 0	13 0	14 0	15 0
16 +	17 +	18 ++	19 ++	20 ++	21 +++	22	23
24	25	26	27	28	29	30	31

Body weight changes: Kg.

0 32.7	7 36.8	14 37.2	21 40	28

Cause of death: -

Post-mortem findings: -

Comments: Lacrimation.

Calf No. 3 Assigned group: Treatment Raw Milk

Admission Date: 7.2.1979

Body Wt. (Kg) on admission: 31.8

Zinc Sulphate Turbidity units: 22

LBC Dosage: 150mg.-feed-twice daily

Duration of administration: 21 days

Survival Time: 21 days

Observation Period: 21 days

Diarrhoea days:

0 0	1 0	2 0	3 0	4 +++	5 +++	6 +++	7 +
8 +++	9 0	10 0	11 0	12 0	13 0	14 0	15 0
16 0	17 0	18 0	19 0	20 0	21 0	22	23
24	25	26	27	28	29	30	31

Body weight changes: Kg.

0	7	14	21	28
31.8	35.9	40	46.3	

Cause of death: -

Post-mortem findings: -

Comments: Umbilical swelling, lacrimation, dull.

Calf No. 4 Assigned group: Treatment Raw Milk

Admission Date: 7.2.1979

Body Wt. (Kg) on admission: 36.3

Zinc Sulphate Turbidity units: 20

LBC Dosage: 150mg.-feed-twice daily

Duration of administration: 21 days

Survival Time: 21 days

Observation Period: 21 days

Diarrhoea days:

0 0	1 0	2 0	3 0	4 0	5 0	6 0	7 0
8 +++	9 +	10 0	11 0	12 0	13 0	14 0	15 0
16 0	17 0	18 0	19 0	20 0	21 0	22	23
24	25	26	27	28	29	30	31

Body weight changes: Kg.

0	7	14	21	28
36.3	40	45	48.1	

Cause of death: -

Post-mortem findings: -

Comments: Dull.

Calf No. 5 Assigned group: Treatment Raw Milk

Admission Date: 7.2.1979

Body Wt. (Kg) on admission: 28.1

Zinc Sulphate Turbidity units: 16

LBC Dosage: 150mg.-feed-twice daily

Duration of administration: 21 days

Survival Time: 8 days

Observation Period: 21 days

Diarrhoea days:

0 0	1 0	2 0	3 0	4 +	5 +	6 +++	7 +++
8 D	9	10	11	12	13	14	15
16	17	18	19	20	21	22	23
24	25	26	27	28	29	30	31

Body weight changes: Kg.

0 28.1	7	14	21	28

Cause of death: Pneumonia

Post-mortem findings: Abomasum-normal. Intestine-congestion. Lung-congestion. No fluid in joints.

Comments: Dull, loss of appetite.

Calf No. 6 Assigned group: Treatment Raw Milk

Admission Date: 7.2.1979

Body Wt. (Kg) on admission: 31.8

Zinc Sulphate Turbidity units: 14

LBC Dosage: 150mg.-feed-twice daily

Duration of administration: 21 days

Survival Time: 21 days

Observation Period: 21 days

Diarrhoea days:

0 0	1 0	2 0	3 0	4 +++	5 +++	6 +++	7 +++
8 0	9 0	10 0	11 0	12 0	13 0	14 0	15 0
16 0	17 0	18 0	19 0	20 0	21 0	22	23
24	25	26	27	28	29	30	31

Body weight changes: Kg.

0 31.8	7 35.9	14 40	21 46.3	28

Cause of death: -

Post-mortem findings: -

Comments: Nasal discharge, coughing.

Calf No. 7 Assigned group: Treatment Raw Milk

Admission Date: 7.2.1979

Body Wt. (Kg) on admission: 28.1

Zinc Sulphate Turbidity units: 16

LBC Dosage: 150mg.-feed-twice daily

Duration of administration: 21 days

Survival Time: 8 days

Observation Period: 21 days

Diarrhoea days:

0 0	1 0	2 0	3 0	4 B	5 +++	6 +++	7 +++
8 D	9	10	11	12	13	14	15
16	17	18	19	20	21	22	23
24	25	26	27	28	29	30	31

Body weight changes: Kg.

0	7	14	21	28
31.8				

Cause of death: Diarrhoea

Post-mortem findings: Abomasum-normal. Intestine-congestion. Lung-normal. No fluid in joints.

Comments: Loss of appetite.

Calf No. 8 Assigned group: Treatment Raw Milk

Admission Date: 7.2.1979

Body Wt. (Kg) on admission: 36.3

Zinc Sulphate Turbidity units: 11

LBC Dosage: 150mg.-feed-twice daily

Duration of administration: 21 days

Survival Time: 8 days

Observation Period: 21 days

Diarrhoea days:

0 0	1 ++	2 B	3 +++	4 +	5 ++	6 ++	7 ++
8 D	9	10	11	12	13	14	15
16	17	18	19	20	21	22	23
24	25	26	27	28	29	30	31

Body weight changes: Kg.

0	7	14	21	28
36.3				

Cause of death: Pneumonia

Post-mortem findings: Abomasum-normal. Intestine-congestion Lung-severe congestion. No fluid in joints.

Comments:
Coughing, lacrimation, loss of appetite. Recumben

Calf No. 9 Assigned group: Treatment Raw Milk

Admission Date: 7.2.1979

Body Wt. (Kg) on admission: 35.4

Zinc Sulphate Turbidity units: 8

LBC Dosage: 150mg.-feed-twice daily

Duration of administration: 21 days

Survival Time: 21 days

Observation Period: 21 days

Diarrhoea days:

0 0	1 ++	2 +++	3 ++	4 +	5 +	6 +	7 +
8 +++	9 +	10 +	11 +	12 ++	13 0	14 +	15 +
16 +	17 +	18 0	19 0	20 0	21 +++	22	23
24	25	26	27	28	29	30	31

Body weight changes: Kg.

0	7	14	21	28
35.4	35.9	40	40	

Cause of death: -

Post-mortem findings: -

Comments: Nasal discharge, lacrimation.

Calf No. 10 Assigned group: Treatment Raw Milk

Admission Date: 7.2.1979

Body Wt. (Kg) on admission: 40

Zinc Sulphate Turbidity units: 7

LBC Dosage: 150mg.-feed-twice daily

Duration of administration: 21 days

Survival Time: 21 days

Observation Period: 21 days

Diarrhoea days:

0 0	1 0	2 0	3 0	4 0	5 0	6 0	7 0
8 0	9 0	10 0	11 0	12 0	13 0	14 0	15 0
16 0	17 0	18 0	19 0	20 0	21	22	23
24	25	26	27	28	29	30	31

Body weight changes: Kg.

0	7	14	21	28
40	45	47.7	55.4	

Cause of death: -

Post-mortem findings: -

Comments: Nasal discharge, coughing.

Calf No. 11 Assigned group: Control Raw Milk

Admission Date: 7.2.1979

Body Wt. (Kg) on admission: 38.1

Zinc Sulphate Turbidity units: 28

LBC Dosage: -

Duration of administration: -

Survival Time: 21 days

Observation Period: 21 days

Diarrhoea days:

0 0	1 0	2 0	3 0	4 0	5 0	6 0	7 0
8 +++	9 +	10 0	11 0	12 +	13 0	14 0	15 0
16 0	17 0	18 0	19 0	20 0	21 0	22	23
24	25	26	27	28	29	30	31

Body weight changes: Kg.

0 38.1	7 40.4	14 43.6	21 48.1	28

Cause of death: -

Post-mortem findings: -

Comments: Salivation.

Calf No. 12 Assigned group: Control Raw Milk

Admission Date: 7.2.1979

Body Wt. (Kg) on admission: 33.6

Zinc Sulphate Turbidity units: 26

LBC Dosage: -

Duration of administration: -

Survival Time: 21 days

Observation Period: 21 days

Diarrhoea days:

0 0	1 0	2 0	3 +	4 0	5 0	6 0	7 0
8 ++	9 0	10 0	11 0	12 0	13 0	14 0	15 0
16 0	17 0	18 0	19 0	20 0	21 0	22	23
24	25	26	27	28	29	30	31

Body weight changes: Kg.

0 33.6	7 36.8	14 39.5	21 45.4	28

Cause of death: -

Post-mortem findings: -

Comments: Coughing.

Calf No. 13 Assigned group: Control Raw Milk

Admission Date: 7.2.1979

Body Wt. (Kg) on admission: 33.6

Zinc Sulphate Turbidity units: 21

LBC Dosage: -

Duration of administration: -

Survival Time: 7 days

Observation Period: 21 days

Diarrhoea days:

0 0	1 0	2 0	3 0	4 0	5 +++	6 +++	7 D
8	9	10	11	12	13	14	15
16	17	18	19	20	21	22	23
24	25	26	27	28	29	30	31

Body weight changes: Kg.

0 33.6	7	14	21	28

Cause of death: Pneumonia

Post-mortem findings: Abomasum-normal. Intestine-congestion. Lung-congestion. No fluid in joints.

Comments: Lacrimation.

Calf No. 14 Assigned group: Control Raw Milk

Admission Date: 7.2.1979

Body Wt. (Kg) on admission: 32.2

Zinc Sulphate Turbidity units: 20

LBC Dosage: -

Duration of administration: -

Survival Time: 6 days

Observation Period: 21 days

Diarrhoea days:

0 0	1 0	2 0	3 +++	4 +++	5 +++	6 D	7
8	9	10	11	12	13	14	15
16	17	18	19	20	21	22	23
24	25	26	27	28	29	30	31

Body weight changes: Kg.

0 32.2	7	14	21	28

Cause of death: Bloat and diarrhoea

Post-mortem findings: Roughage in abomasum. Intestine-congestion. Lung-normal. No fluid in joints.

Comments: Recumbent before death - lacrimation.

Calf No. 15 Assigned group: Control Raw Milk

Admission Date: 7.2.1979

Body Wt. (Kg) on admission: 31.8

Zinc Sulphate Turbidity units: 18

LBC Dosage: -

Duration of administration: -

Survival Time: 7 days

Observation Period: 21 days

Diarrhoea days:

0 0	1 0	2 +++	3 +++	4 +++	5 +++	6 +++	7 D
8	9	10	11	12	13	14	15
16	17	18	19	20	21	22	23
24	25	26	27	28	29	30	31

Body weight changes: Kg.

0	7	14	21	28
31.8				

Cause of death: Diarrhoea

Post-mortem findings: Abomasum-normal. Intestine-congestion. Lung-normal. No fluid in joints.

Comments: Lacrimation.

Calf No. 16 Assigned group: Control Raw Milk

Admission Date: 7.2.1979

Body Wt. (Kg) on admission: 39

Zinc Sulphate Turbidity units: 13

LBC Dosage: -

Duration of administration: -

Survival Time: 21 days

Observation Period: 21 days

Diarrhoea days:

0 0	1 0	2 +++	3 B	4 +++	5 ++	6 +	7 +
8 0	9 +++	10 0	11 0	12 ++	13 0	14 0	15 0
16 0	17 0	18 0	19 0	20 0	21 0	22	23
24	25	26	27	28	29	30	31

Body weight changes: Kg.

0	7	14	21	28
39	39.5	40.9	45.4	

Cause of death: -

Post-mortem findings: -

Comments:

Calf No. 17 Assigned group: Control Raw Milk

Admission Date: 7.2.1979

Body Wt. (Kg) on admission: 29

Zinc Sulphate Turbidity units: 12

LBC Dosage: -

Duration of administration: -

Survival Time: 6 days

Observation Period: 21 days

Diarrhoea days:

0 0	1 0	2 0	3 0	4 +++	5 +++	6 D	7
8	9	10	11	12	13	14	15
16	17	18	19	20	21	22	23
24	25	26	27	28	29	30	31

Body weight changes: Kg.

0 29	7	14	21	28

Cause of death: Diarrhoea

Post-mortem findings: Abomasum-normal. Intestine-congestion. Lung-normal. No fluid in joints.

Comments: Lacrimation.

Calf No. 18 Assigned group: Control Raw Milk

Admission Date: 7.2.1979

Body Wt. (Kg) on admission: 35.9

Zinc Sulphate Turbidity units: 12

LBC Dosage: -

Duration of administration: -

Survival Time: 21 days

Observation Period: 21 days

Diarrhoea days:

0 0	1 0	2 0	3 +	4 ++	5 +++	6 +++	7 +++
8 +++	9 +++	10 +	11 0	12 0	13 0	14 0	15 0
16 0	17 0	18 0	19 0	20 0	21 0	22	23
24	25	26	27	28	29	30	31

Body weight changes: Kg.

0 35.9	7 39.5	14 41.3	21 47.7	28

Cause of death: -

Post-mortem findings: -

Comments: Umbilical swelling

Calf No. 19 Assigned group: Control Raw Milk

Admission Date: 7.2.1979

Body Wt. (Kg) on admission: 33.6

Zinc Sulphate Turbidity units: 8

LBC Dosage: -

Duration of administration: -

Survival Time: 7 days

Observation Period: 21 days

Diarrhoea days:

0 0	1 0	2 0	3 +++	4 B	5 B	6 +++	7 D
8	9	10	11	12	13	14	15
16	17	18	19	20	21	22	23
24	25	26	27	28	29	30	31

Body weight changes: Kg.

0	7	14	21	28
33.6				

Cause of death: Diarrhoea

Post-mortem findings: Abomasum-normal. Intestine-severe congestion. Lung-normal. No fluid in joints.

Comments:

Calf No.20 Assigned group: Control Raw Milk

Admission Date: 7.2.1979

Body Wt. (Kg) on admission: 36.3

Zinc Sulphate Turbidity units: 7

LBC Dosage: -

Duration of administration: -

Survival Time: 21 days

Observation Period: 21 days

Diarrhoea days:

0 0	1 0	2 0	3 0	4 ++	5 +++	6 +++	7 +++
8 +++	9 +	10 0	11 0	12 0	13 0	14 0	15 0
16 0	17 0	18 0	19 0	20 0	21 0	22	23
24	25	26	27	28	29	30	31

Body weight changes: Kg.

0	7	14	21	28
36.3	39.5	41.3	47.7	

Cause of death: -

Post-mortem findings: -

Comments:

APPENDIX 4 : SUMMARY OF DATA REGARDING SOURCE, ZST VALUES, WEIGHT CHANGES, GROUP ALLOCATION AND TREATMENT OF 20 INDIVIDUAL CALVES STUDIED IN EXPERIMENT 2.

EXPERIMENT 2

TREATMENT		75mgm LBC twice daily in feed; 14 days					CONTROL						
Calf No.	Market	ZST	Bwt. (Kg) 21-3	Bwt. (Kg) 28-3	Bwt. (Kg) 4-4	Weight Gain	Calf No.	Market	ZST	Bwt. (Kg) 21-3	Bwt. (Kg) 28-3	Bwt. (Kg) 4-4	Weight Gain
High							High						
21	Lanark	32	35	-	-	-	31	Lanark	38	44	42	-	-
22	Ayr	29	40	41	44	+4	32	Paisley	35	40	-	-	-
23	Ayr	26	36	40	40	+4	33	Paisley	22	35	30	-	-
Mean		29.0	37.0	40.5	42.0		Mean		31.7	39.7	36.0	-	-
Intermediate							Intermediate						
24	Paisley	22	36	-	-	-	34	Paisley	18	37	-	-	-
25	Lanark	17	38	38	37	-1	35	Paisley	14	36	37	38	+2
26	Ayr	14	36	-	-	-	36	Ayr	11	34	32	33	-1
27	Lanark	11	44	42	45	+1	37	Paisley	9	42	40	40	-2
Mean		16.0	38.5	40.0	41.0		Mean		13	37.3	36.3	37.0	
Low							Low						
28	Paisley	8	34	33	32	-2	38	Paisley	9	40	-	-	-
29	Paisley	7	33	-	-	-	39	Paisley	8	33	36	37	+4
30	Paisley	5	45	-	-	-	40	Paisley	7	36	-	-	-
Mean		6.7	37.3	33.0	32.0		Mean		8	36.3	36.0	37.0	
MEAN		17.1	37.7	38.8	39.6	1.2	MEAN		17.1	37.7	36.2	37.0	0.75

APPENDIX 5 (TABLES 21-40) : INDIVIDUAL DATA SHEETS FOR 20 CALVES USED IN EXPERIMENT 2, INCLUDING INFORMATION REGARDING PRESENCE (AND DEGREE) OR ABSENCE OF DIARRHOEA, SURVIVAL TIME AND GROSS POST MORTEM FINDINGS WHERE APPLICABLE.

Calf No. 21 Assigned group: Treatment Raw Milk

Admission Date: 21.3.1979

Body Wt. (Kg) on admission: 35

Zinc Sulphate Turbidity units: 32

LBC Dosage: 75mg.-feed-twice daily

Duration of administration: 14 days

Survival Time: 5 days

Observation Period: 14 days

Diarrhoea days:

0 0	1 0	2 0	3 +++	4 +++	5 D	6	7
8	9	10	11	12	13	14	15
16	17	18	19	20	21	22	23
24	25	26	27	28	29	30	31

Body weight changes: Kg.

0 35	7	14	21	28

Cause of death: Pneumonia and diarrhoea

Post-mortem findings: Abomasum-erosion. Intestine-congestion and full of air. Lung-congestion. No fluid in joints.

Comments: Coughing.

Calf No. 22 Assigned group: Treatment Raw Milk

Admission Date: 21.3.1979

Body Wt. (Kg) on admission: 40

Zinc Sulphate Turbidity units: 29

LBC Dosage: 75mg.-feed-twice daily

Duration of administration: 14 days

Survival Time: 14 days

Observation Period: 14 days

Diarrhoea days:

0 0	1 0	2 0	3 0	4 0	5 ++	6 +++	7 ++
8 0	9 0	10 0	11 0	12 0	13 0	14 0	15
16	17	18	19	20	21	22	23
24	25	26	27	28	29	30	31

Body weight changes: Kg.

0 40	7 41	14 44	21	28

Cause of death: -

Post-mortem findings: -

Comments:

Calf No. 23 Assigned group: Treatment Raw Milk

Admission Date: 21.3.1979

Body Wt. (Kg) on admission: 36

Zinc Sulphate Turbidity units: 26

LBC Dosage: 75mg.-feed-twice daily

Duration of administration: 14 days

Survival Time: 14 days

Observation Period: 14 days

Diarrhoea days:

0 0	1 0	2 0	3 0	4 0	5 0	6 0	7 +++
8 +++	9 0	10 0	11 0	12 0	13 0	14 0	15
16	17	18	19	20	21	22	23
24	25	26	27	28	29	30	31

Body weight changes: Kg.

0	7	14	21	28
36.	40	40		

Cause of death: -

Post-mortem findings: -

Comments:

Calf No. 24 Assigned group: Treatment Raw Milk

Admission Date: 21.3.1979

Body Wt. (Kg) on admission: 36

Zinc Sulphate Turbidity units: 22

LBC Dosage: 75mg.-feed-twice daily

Duration of administration: 14 days

Survival Time: 8 days

Observation Period: 14 days

Diarrhoea days:

0 0	1 0	2 0	3 0	4 +++	5 +++	6 +++	7 +++
8 D	9	10	11	12	13	14	15
16	17	18	19	20	21	22	23
24	25	26	27	28	29	30	31

Body weight changes: Kg.

0	7	14	21	28
36				

Cause of death: Pneumonia and diarrhoea

Post-mortem findings: Abomasum-erosion. Intestine-congestio and full of air. Lung-congestion. No fluid in joints.

Comments:
Coughing.

Calf No. 25 Assigned group: Treatment Raw Milk

Admission Date: 21.3.1979

Body Wt. (Kg) on admission: 38

Zinc Sulphate Turbidity units: 17

LBC Dosage: 75mg.-feed-twice daily

Duration of administration: 14 days

Survival Time: 14 days

Observation Period: 14 days

Diarrhoea days:

0 0	1 0	2 0	3 0	4 0	5 0	6 0	7 +++
8 +++	9 0	10 0	11 ++	12 0	13 0	14 0	15
16	17	18	19	20	21	22	23
24	25	26	27	28	29	30	31

Body weight changes: Kg.

0 38	7 38	14 37	21	28

Cause of death: -

Post-mortem findings: -

Comments: Nasal discharge, lacrimation. Coughing.

Calf No. 26 Assigned group: Treatment Raw Milk

Admission Date: 21.3.1979

Body Wt. (Kg) on admission: 36

Zinc Sulphate Turbidity units: 14

LBC Dosage: 75mg.-feed-twice daily

Duration of administration: 14 days

Survival Time: 5 days

Observation Period: 14 days

Diarrhoea days:

0 0	1 0	2 0	3 +++	4 +++	5 D	6	7
8	9	10	11	12	13	14	15
16	17	18	19	20	21	22	23
24	25	26	27	28	29	30	31

Body weight changes: Kg.

0 36	7	14	21	28

Cause of death: Diarrhoea

Post-mortem findings: Abomasum-erosion. Intestine-congestio
and full of air. Lung-normal. No fluid in joints.
Comments:

Calf No. 27 Assigned group: Treatment Raw Milk

Admission Date: 21.3.1979

Body Wt. (Kg) on admission: 44

Zinc Sulphate Turbidity units: 11

LBC Dosage: 75mg.-feed-twice daily

Duration of administration: 14 days

Survival Time: 14 days

Observation Period: 14 days

Diarrhoea days:

0 0	1 0	2 +++	3 +	4 0	5 +++	6 +++	7 ++
8 0	9 +	10 0	11 0	12 0	13 0	14 0	15
16	17	18	19	20	21	22	23
24	25	26	27	28	29	30	31

Body weight changes: Kg.

0 44	7 42	14 45	21	28

Cause of death: -

Post-mortem findings: -

Comments:

Calf No. 28 Assigned group: Treatment Raw Milk

Admission Date: 21.3.1979

Body Wt. (Kg) on admission: 34

Zinc Sulphate Turbidity units: 8

LBC Dosage: 75mg.-feed-twice daily

Duration of administration: 14 days

Survival Time: 14 days

Observation Period: 14 days

Diarrhoea days:

0 0	1 +++	2 +++	3 0	4 ++	5 +++	6 +++	7 ++
8 ++	9 0	10 +	11 0	12 ++	13 ++	14 ++	15
16	17	18	19	20	21	22	23
24	25	26	27	28	29	30	31

Body weight changes: Kg.

0 34	7 33	14 32	21	28

Cause of death: -

Post-mortem findings: -

Comments:

Calf No. 29 Assigned group: Treatment Raw Milk

Admission Date: 21.3.1979

Body Wt. (Kg) on admission: 33

Zinc Sulphate Turbidity units: 7

LBC Dosage: 75mg.-feed-twice daily

Duration of administration: 14 days

Survival Time: 4 days

Observation Period: 14 days

Diarrhoea days:

0 0	1 +++	2 +++	3 +	4 D	5	6	7
8	9	10	11	12	13	14	15
16	17	18	19	20	21	22	23
24	25	26	27	28	29	30	31

Body weight changes: Kg.

0 33	7	14	21	28

Cause of death: Diarrhoea

Post-mortem findings: Abomasum-normal. Intestine-slight congestion.Lung-normal. No fluid in joints.

Comments:

Calf No. 30 Assigned group: Treatment Raw Milk

Admission Date: 21.3.1979

Body Wt. (Kg) on admission: 45

Zinc Sulphate Turbidity units: 5

LBC Dosage: 75mg.-feed-twice daily

Duration of administration: 14 days

Survival Time: 6 days

Observation Period: 14 days

Diarrhoea days:

0 0	1 0	2 0	3 0	4 ++	5 +++	6 D	7
8	9	10	11	12	13	14	15
16	17	18	19	20	21	22	23
24	25	26	27	28	29	30	31

Body weight changes: Kg.

0 45	7	14	21	28

Cause of death: Diarrhoea

Post-mortem findings: Abomasum-erosion. Intestine-congestion and full of air. Lung-normal. No fluid in joints.

Comments:

Calf No. 31 Assigned group: Control Raw Milk

Admission Date: 21.3.1979

Body Wt. (Kg) on admission: 44

Zinc Sulphate Turbidity units: 38

LBC Dosage: -

Duration of administration: -

Survival Time: 10 days

Observation Period: 14 days

Diarrhoea days:

0 0	1 0	2 0	3 0	4 0	5 +++	6 +++	7 +++
8 ++	9 +++	10 D	11	12	13	14	15
16	17	18	19	20	21	22	23
24	25	26	27	28	29	30	31

Body weight changes: Kg.

0	7	14	21	28

Cause of death: Diarrhoea and pneumonia

Post-mortem findings: Abomasum-congestion. Intestine-congestion and full of air. Lung-congestion. No fluid in joints.

Comments: Coughing

Calf No. 32 Assigned group: Control Raw Milk

Admission Date: 21.3.1979

Body Wt. (Kg) on admission: 40

Zinc Sulphate Turbidity units: 35

LBC Dosage: -

Duration of administration: -

Survival Time: 6 days

Observation Period: 14 days

Diarrhoea days:

0 0	1 0	2 0	3 0	4 0	5 +++	6 D	7
8	9	10	11	12	13	14	15
16	17	18	19	20	21	22	23
24	25	26	27	28	29	30	31

Body weight changes: Kg.

0	7	14	21	28

Cause of death: Diarrhoea

Post-mortem findings: Abomasum-congestion. Intestine-congestion and full of air. Lung-normal. No fluid in joints

Comments:

Calf No. 33 Assigned group: Control Raw Milk

Admission Date: 21.3.1979

Body Wt. (Kg) on admission: 35

Zinc Sulphate Turbidity units: 22

LBC Dosage: -

Duration of administration: -

Survival Time: 9 days

Observation Period: 14 days

Diarrhoea days:

0 0	1 0	2 +	3 B	4 +++	5 +++	6 +++	7 +++
8 0	9 D	10	11	12	13	14	15
16	17	18	19	20	21	22	23
24	25	26	27	28	29	30	31

Body weight changes: Kg.

0	7	14	21	28

Cause of death: Diarrhoea

Post-mortem findings: Abomasum-normal. Intestine-congestion and full of air. Lung-normal. No fluid in joints.
Comments:

Calf No. 34 Assigned group: Control Raw Milk

Admission Date: 21.3.1979

Body Wt. (Kg) on admission: 37

Zinc Sulphate Turbidity units: 18

LBC Dosage: -

Duration of administration: -

Survival Time: 7 days

Observation Period: 14 days

Diarrhoea days:

0 0	1 0	2 0	3 +++	4 +++	5 +++	6 +++	7 D
8	9	10	11	12	13	14	15
16	17	18	19	20	21	22	23
24	25	26	27	28	29	30	31

Body weight changes: Kg.

0	7	14	21	28

Cause of death: Diarrhoea

Post-mortem findings: Abomasum-erosion. Intestine-congestion and full of air. Lung-normal. No fluid in joints.
Comments:

Calf No. 35 Assigned group: Control Raw Milk

Admission Date: 21.3.1979

Body Wt. (Kg) on admission: 36

Zinc Sulphate Turbidity units: 14

LBC Dosage: -

Duration of administration: -

Survival Time: 14 days

Observation Period: 14 days

Diarrhoea days:

0 0	1 +	2 +++	3 0	4 0	5 +++	6 0	7 +++
8 0	9 0	10 ++	11 0	12 0	13 0	14 0	15
16	17	18	19	20	21	22	23
24	25	26	27	28	29	30	31

Body weight changes: Kg.

0 36	7 37	14 38	21	28

Cause of death: -

Post-mortem findings: -

Comments:

Calf No. 36 Assigned group: Control Raw Milk

Admission Date: 21.3.1979

Body Wt. (Kg) on admission: 34

Zinc Sulphate Turbidity units: 11

LBC Dosage: -

Duration of administration: -

Survival Time: 14 days

Observation Period: 14 days

Diarrhoea days:

0 0	1 ++	2 +++	3 +++	4 +++	5 +++	6 ++	7 +++
8 +++	9 +	10 +++	11 0	12 0	13 0	14 0	15
16	17	18	19	20	21	22	23
24	25	26	27	28	29	30	31

Body weight changes: Kg.

0 34	7 32	14 33	21	28

Cause of death: -

Post-mortem findings: -

Comments:

Calf No. 37 Assigned group: Control Raw Milk

Admission Date: 21.3.1979

Body Wt. (Kg) on admission: 42

Zinc Sulphate Turbidity units: 9

LBC Dosage: -

Duration of administration: -

Survival Time: 14 days

Observation Period: 14 days

Diarrhoea days:

0 0	1 +++	2 +	3 0	4 +++	5 +++	6 +++	7 +++
8 +++	9 +++	10 ++	11 +	12 0	13 0	14 0	15
16	17	18	19	20	21	22	23
24	25	26	27	28	29	30	31

Body weight changes: Kg.

0 42	7 40	14 40	21	28

Cause of death: -

Post-mortem findings: -

Comments:

Calf No. 38 Assigned group: Control Raw Milk

Admission Date: 21.3.1979

Body Wt. (Kg) on admission: 40

Zinc Sulphate Turbidity units: 9

LBC Dosage: -

Duration of administration: -

Survival Time: 4 days

Observation Period: 14 days

Diarrhoea days:

0 0	1 0	2 +++	3 +++	4 D	5	6	7
8	9	10	11	12	13	14	15
16	17	18	19	20	21	22	23
24	25	26	27	28	29	30	31

Body weight changes: Kg.

0 40	7	14	21	28

Cause of death: Diarrhoea

Post-mortem findings: Abomasum-erosion. Intestine-congestion and full of air. Lung-normal. No fluid in joints.

Comments:

Calf No. 39 Assigned group: Control Raw Milk

Admission Date: 21.3.1979

Body Wt. (Kg) on admission: 33

Zinc Sulphate Turbidity units: 8

LBC Dosage: -

Duration of administration: -

Survival Time: 14 days

Observation Period: 14 days

Diarrhoea days:

0 0	1 +++	2 +	3 +++	4 ++	5 ++	6 0	7 0
8 +	9 0	10 0	11 0	12 0	13 0	14 0	15
16	17	18	19	20	21	22	23
24	25	26	27	28	29	30	31

Body weight changes: Kg.

0 33	7 36	14 37	21	28

Cause of death: -

Post-mortem findings: -

Comments:

Calf No. 40 Assigned group: Control Raw Milk

Admission Date: 21.3.1979

Body Wt. (Kg) on admission: 36

Zinc Sulphate Turbidity units: 7

LBC Dosage: -

Duration of administration: -

Survival Time: 7 days

Observation Period: 14 days

Diarrhoea days:

0 0	1 0	2 0	3 0	4 +	5 +++	6 +++	7 D
8	9	10	11	12	13	14	15
16	17	18	19	20	21	22	23
24	25	26	27	28	29	30	31

Body weight changes: Kg.

0 36	7	14	21	28

Cause of death: Pneumonia and diarrhoea

Post-mortem findings: Abomasum-erosion. Intestine-congestion and full of air. Lung-congestion. No fluid in joints.

Comments: Coughing.

APPENDIX 6 : SUMMARY OF DATA REGARDING SOURCE, ZST VALUES, WEIGHT CHANGES, GROUP ALLOCATION AND TREATMENT OF 20 INDIVIDUAL CALVES STUDIED IN EXPERIMENT 3

EXPERIMENT 3

TREATMENT		150mgm LBC twice daily in feed; 7 days 75mgm LBC twice daily in feed; 7 days					CONTROL						
Calf No.	Market	ZST	Bwt. (Kg) 19-4	Bwt. (Kg) 25-4	Bwt. (Kg) 2-5	Weight Gain	Calf No.	Market	ZST	Bwt. (Kg) 19-4	Bwt. (Kg) 25-4	Bwt. (Kg) 2-5	Weight Gain
High							High						
41	Ayr	22	35	-	-	-	51	Ayr	24	26	-	-	-
42	Ayr	15	41	44	46	+5	52	Strathaven	14	41	38	36	-5
43	Strathaven	11	35	-	-	-	53	Ayr	11	30	-	-	-
Mean		16.0	37.0	44.0	46.0		Mean		16.3	32.3	38.0	36.0	
Intermediate							Intermediate						
44	Ayr	10	33	-	-	-	54	Strathaven	11	46	45	48	+2
45	Strathaven	10	34	33	33	-1	55	Ayr	10	42	39	41	-1
46	Ayr	8	37	-	-	-	56	Strathaven	8	40	41	47	+7
47	Strathaven	7	34	-	-	-	57	Strathaven	4	31	25	-	-
Mean		8.8	34.5	33.0	33.0		Mean		8.3	39.8	37.5	45.3	
Low							Low						
48	Strathaven	3	32	32	30	-2	58	Strathaven	3	37	-	-	-
49	Strathaven	2	40	37	-	-	59	Ayr	3	41	-	-	-
50	Strathaven	2	41	-	-	-	60	Strathaven	2	38	-	-	-
Mean		2.3	37.7	34.5	30.0		Mean		2.7	38.7	-	-	
MEAN		9	36.2	36.5	36.3	0.5	MEAN		9.0	37.2	37.6	43.0	0.75

APPENDIX 7 (TABLES 41-60) : INDIVIDUAL DATA SHEETS FOR 20 CALVES USED IN EXPERIMENT 3, INCLUDING INFORMATION REGARDING PRESENCE (AND DEGREE) OR ABSENCE OF DIARRHOEA, SURVIVAL TIME AND GROSS POST MORTEM FINDINGS WHERE APPLICABLE.

Calf No. 41 Assigned group: Treatment Raw Milk

Admission Date: 19.4.1979

Body Wt. (Kg) on admission: 35

Zinc Sulphate Turbidity units: 22

LBC Dosage: 150mg.-feed-twice daily-7 days, then 75mg.-feed-twice daily-7 days.

Duration of administration:

Survival Time: 7 days

Observation Period: 14 days

Diarrhoea days:

0 0	1 0	2 0	3 +++	4 +++	5 ++	6 +++	7 D
8	9	10	11	12	13	14	15
16	17	18	19	20	21	22	23
24	25	26	27	28	29	30	31

Body weight changes: Kg.

0 35	7	14	21	28

Cause of death: Diarrhoea

Post-mortem findings: Abomasum-congestion. Intestine-congestion and full of air. Lung-normal. No fluid in joints.

Comments: Umbilical cord inflamed and full of pus.

Calf No. 42 Assigned group: Treatment Raw Milk

Admission Date: 19.4.1979

Body Wt. (Kg) on admission: 41

Zinc Sulphate Turbidity units: 15

LBC Dosage: 150mg.-feed-twice daily-7 days, then 75mg.-feed-twice daily-7 days.

Duration of administration:

Survival Time: 14 days

Observation Period: 14 days

Diarrhoea days:

0 0	1 +++	2 +	3 0	4 0	5 0	6 +++	7 0
8 0	9 0	10 +	11 0	12 0	13 0	14 0	15
16	17	18	19	20	21	22	23
24	25	26	27	28	29	30	31

Body weight changes: Kg.

0 41	7 44	14 46	21	28

Cause of death: -

Post-mortem findings: -

Comments:

Calf No. 43 Assigned group: Treatment Raw Milk

Admission Date: 19.4.1979

Body Wt. (Kg) on admission: 35

Zinc Sulphate Turbidity units: 11

LBC Dosage: 150mg.-feed-twice daily-7 days, then 75mg.-feed-twice daily-7 days.

Duration of administration:

Survival Time: 6 days

Observation Period: 14 days

Diarrhoea days:

0 0	1 0	2 +	3 +++	14 +++	5 +++	6 D	7
8	9	10	11	12	13	14	15
16	17	18	19	20	21	22	23
24	25	26	27	28	29	30	31

Body weight changes:

0 35	7	14	21	28

Cause of death: Diarrhoea

Post-mortem findings: Abomasum-congestion. Intestine-congestion and full of air. Lung-normal. No fluid in joints.

Comments:
Umbilical cord inflamed and full of pus.

Calf No. 44 Assigned group: Treatment Raw Milk

Admission Date: 19.3.1979

Body Wt. (Kg) on admission: 33

Zinc Sulphate Turbidity units: 10

LBC Dosage: 150mg.-feed-twice daily-7 days, then 75mg.-feed-twice daily-7 days.

Duration of administration:

Survival Time: 5 days

Observation Period: 14 days

Diarrhoea days:

0 0	1 +	2 +++	3 +++	4 +++	5 D	6	7
8	9	10	11	12	13	14	15
16	17	18	19	20	21	22	23
24	25	26	27	28	29	30	31

Body weight changes:

0 33	7	14	21	28

Cause of death: Diarrhoea and pneumonia.

Post-mortem findings: Abomasum-congestion. Intestine-congestion and full of air. Lung-congestion. Excessive fluid in joints.

Comments:
Umbilical cord inflamed.
Dull and recumbent before death.

Calf No. 45 Assigned group: Treatment Raw Milk

Admission Date: 19.4.1979

Body Wt. (Kg) on admission: 34

Zinc Sulphate Turbidity units: 10

LBC Dosage: 150mg.-feed-twice daily-7 days, then 75mg.-feed-twice daily-7 days.

Duration of administration:

Survival Time: 14 days

Observation Period: 14 days

Diarrhoea days:

0 0	1 0	2 +++	3 0	4 +++	5 +++	6 +++	7 0
8 +++	9 +	10 +	11 0	12 0	13 0	14 0	15
16	17	18	19	20	21	22	23
24	25	26	27	28	29	30	31

Body weight changes: Kg.

0 34	7 33	14 33	21	28

Cause of death: -

Post-mortem findings: -

Comments:

Calf No. 46 Assigned group: Treatment Raw Milk

Admission Date: 19.4.1979

Body Wt. (Kg) on admission: 37

Zinc Sulphate Turbidity units: 8

LBC Dosage: 150mg.-feed-twice daily-7 days, then 75mg.-feed-twice daily-7 days.

Duration of administration:

Survival Time: 5 days

Observation Period: 14 days

Diarrhoea days:

0 0	1 0	2 +++	3 +++	4 +++	5 D	6	7
8	9	10	11	12	13	14	15
16	17	18	19	20	21	22	23
24	25	26	27	28	29	30	31

Body weight changes: Kg.

0 37	7	14	21	28

Cause of death: Diarrhoea

Post-mortem findings: Abomasum-congestion. Intestine-congested and full of air. Lung-normal. No fluid in joints.

Comments: Umbilical cord inflamed and full of pus. Dull and signs of meningitis before death.

Calf No. 47 Assigned group: Treatment Raw Milk

Admission Date: 19.4.1979

Body Wt. (Kg) on admission: 34

Zinc Sulphate Turbidity units: 7

LBC Dosage: 150mg.-feed-twice daily-7 days, then 75mg.-feed-twice daily-7 days.

Duration of administration:

Survival Time: 5 days

Observation Period: 14 days

Diarrhoea days:

0 0	1 +++	2 +++	3 +++	4 +++	5 0	6	7
8	9	10	11	12	13	14	15
16	17	18	19	20	21	22	23
24	25	26	27	28	29	30	31

Body weight changes: Kg.

0 34	7	14	21	28

Cause of death: Diarrhoea and Pneumonia

Post-mortem findings: Abomasum-erosion, Intestine-congestion and full of air. Lung-congestion. No fluid in joints.

Comments: Umbilical cord inflamed and full of pus. Dull and recumbent before death.

Calf No. 48 Assigned group: Treatment Raw Milk

Admission Date: 19.4.1979

Body Wt. (Kg) on admission: 32

Zinc Sulphate Turbidity units: 3

LBC Dosage: 150mg.-feed-twice daily-7 days, then 75mg.-feed-twice daily-7 days.

Duration of administration:

Survival Time: 14 days

Observation Period: 14 days

Diarrhoea days:

0 0	1 0	2 0	3 0	4 0	5 +++	6 B	7 ++
8 0	9 0	10 0	11 +++	12 0	13 0	14 0	15
16	17	18	19	20	21	22	23
24	25	26	27	28	29	30	31

Body weight changes: Kg.

0 32	7 32	14 30	21	28

Cause of death: -

Post-mortem findings: -

Comments:

Calf No. 49 Assigned group: Treatment Raw Milk

Admission Date: 19.4.1979

Body Wt. (Kg) on admission: 40

Zinc Sulphate Turbidity units: 2

LBC Dosage: 150mg.-feed-twice daily-7 days, then 75mg.-feed-twice daily-7 days.

Duration of administration:

Survival Time: 8 days

Observation Period: 14 days

Diarrhoea days:

0 0	1 0	2 0	3 0	4 +	5 +++	6 B	7 +++
8 D	9	10	11	12	13	14	15
16	17	18	19	20	21	22	23
24	25	26	27	28	29	30	31

Body weight changes: Kg.

0 40	7 37	14	21	28

Cause of death: Diarrhoea and pneumonia

Post-mortem findings: Abomasum-congestion. Intestine-congestion and full of air. Lung-congestion. No fluid in joints.

Comments:
Dull and recumbent before death.

Calf No. 50 Assigned group: Treatment Raw Milk

Admission Date: 19.4.1979

Body Wt. (Kg) on admission: 41

Zinc Sulphate Turbidity units: 2

LBC Dosage: 150mg.-feed-twice daily-7 days, then 75mg.-feed-twice daily-7 days.

Duration of administration:

Survival Time: 3 days

Observation Period: 14 days

Diarrhoea days:

0 0	1 0	2 +++	3 D	4	5	6	7
8	9	10	11	12	13	14	15
16	17	18	19	20	21	22	23
24	25	26	27	28	29	30	31

Body weight changes: Kg.

0 41	7	14	21	28

Cause of death: Diarrhoea

Post-mortem findings: Abomasum-normal. Intestine-congest
and full of air. Lung-normal. Excessive fluid in joints.

Comments:
The umbilical cord inflamed and full of pus.
Dull and recumbent before death.

Calf No. 51 Assigned group: Control Raw Milk

Admission Date: 19.4.1979

Body Wt. (Kg) on admission: 26

Zinc Sulphate Turbidity units: 24

LBC Dosage: -

Duration of administration: -

Survival Time: 7 days

Observation Period: 14 days

Diarrhoea days:

0 0	1 +	2 +++	3 +++	4 +++	5 +++	6 +++	7 D
8	9	10	11	12	13	14	15
16	17	18	19	20	21	22	23
24	25	26	27	28	29	30	31

Body weight changes: Kg.

0 26	7	14	21	28

Cause of death: Diarrhoea and pneumonia

Post-mortem findings: Abomasum-erosion. Intestine-congestion and full of air. Lung-congestion. No fluid in joints.

Comments:
Umbilical cord inflamed and full of pus.

Calf No. 52 Assigned group: Control Raw Milk

Admission Date:19.4.1979

Body Wt. (Kg) on admission: 41

Zinc Sulphate Turbidity units: 14

LBC Dosage: -

Duration of administration: -

Survival Time: 14 days

Observation Period: days

Diarrhoea days:

0 0	1 0	2 0	3 0	4 0	5 ++	6 ++	7 +++
8 +++	9 0	10 0	11 0	12 0	13 0	14 0	15
16	17	18	19	20	21	22	23
24	25	26	27	28	29	30	31

Body weight changes: Kg.

0 41	7 38	14 36	21	28

Cause of death: -

Post-mortem findings: -

Comments:

Calf No. 53 Assigned group: Control Raw Milk

Admission Date: 19.4.1979

Body Wt. (Kg) on admission: 30

Zinc Sulphate Turbidity units: 11

LBC Dosage: -

Duration of administration: -

Survival Time: 6 days

Observation Period: 14 days

Diarrhoea days:

0 0	1 0	2 0	3 0	4 0	5 +++	6 D	7
8	9	10	11	12	13	14	15
16	17	18	19	20	21	22	23
24	25	26	27	28	29	30	31

Body weight changes: Kg.

0 30	7	14	21	28

Cause of death: Diarrhoea and pneumonia

Post-mortem findings: Abomasum-normal. Intestine-congestion and full of air. Lung-congestion. No fluid in joints.

Comments: Umbilical cord inflamed and full of pus.

Calf No. 54 Assigned group: Control Raw Milk

Admission Date: 19.4.1979

Body Wt. (Kg) on admission: 46

Zinc Sulphate Turbidity units: 11

LBC Dosage: -

Duration of administration: -

Survival Time: 14 days

Observation Period: 14 days

Diarrhoea days:

0 0	1 0	2 0	3 0	4 0	5 +++	6 +++	7 0
8 0	9 0	10 0	11 0	12 0	13 0	14 0	15
16	17	18	19	20	21	22	23
24	25	26	27	28	29	30	31

Body weight changes: Kg.

0 46	7 45	14 48	21	28

Cause of death: -

Post-mortem findings: -

Comments:

Calf No. 55 Assigned group: Control Raw Milk

Admission Date: 19.4.1979

Body Wt. (Kg) on admission: 42

Zinc Sulphate Turbidity units: 10

LBC Dosage: -

Duration of administration: -

Survival Time: 14 days

Observation Period: 14 days

Diarrhoea days:

0 0	1 0	2 0	3 +++	4 +++	5 +++	6 ++	7 0
8 0	9 0	10 0	11 0	12 0	13 0	14 0	15
16	17	18	19	20	21	22	23
24	25	26	27	28	29	30	31

Body weight changes: Kg.

0 42	7 39	14 41	21	28

Cause of death: -

Post-mortem findings: -

Comments:

Calf No. 56 Assigned group: Control Raw Milk

Admission Date: 19.4.1979

Body Wt. (Kg) on admission: 40

Zinc Sulphate Turbidity units: 8

LBC Dosage: -

Duration of administration: -

Survival Time: 14 days

Observation Period: 14 days

Diarrhoea days:

0 0	1 0	2 0	3 0	4 +++	5 +++	6 ++	7 0
8 0	9 +++	10 0	11 0	12 0	13 0	14 0	15
16	17	18	19	20	21	22	23
24	25	26	27	28	29	30	31

Body weight changes: Kg.

0 40	7 41	14 47	21	28

Cause of death: -

Post-mortem findings: -

Comments:

Calf No. 57 Assigned group: Control Raw Milk

Admission Date: 19.4.1979

Body Wt. (Kg) on admission: 31

Zinc Sulphate Turbidity units: 4

LBC Dosage: -

Duration of administration: -

Survival Time: 8 days

Observation Period: 14 days

Diarrhoea days:

0 0	1 +++	2 +++	3 +++	4 +++	5 +++	6 +++	7 +++
8 D	9	10	11	12	13	14	15
16	17	18	19	20	21	22	23
24	25	26	27	28	29	30	31

Body weight changes: Kg.

0 31	7 25	14	21	28

Cause of death: Diarrhoea

Post-mortem findings: Abomasum-normal. Intestine-congestion and full of air. Lung-normal. No fluid in joints.

Comments:

Dull and recumbent before death.

Calf No. 58 Assigned group: Control Raw Milk

Admission Date: 19.4.1979

Body Wt. (Kg) on admission: 37

Zinc Sulphate Turbidity units: 3

LBC Dosage: -

Duration of administration: -

Survival Time: 5 days

Observation Period: 14 days

Diarrhoea days:

0 0	1 +	2 +++	3 +++	4 +++	5 D	6	7
8	9	10	11	12	13	14	15
16	17	18	19	20	21	22	23
24	25	26	27	28	29	30	31

Body weight changes: Kg.

0 37	7	14	21	28

Cause of death: Diarrhoea and pneumonia

Post-mortem findings: Abomasum-normal. Intestine-congestion and full of air. Lung-congestion. No fluid in joints.

Comments:

Umbilical cord inflamed and full of pus.

Calf No. 59 Assigned group: Control Raw Milk

Admission Date: 19.4.1979

Body Wt. (Kg) on admission: 41

Zinc Sulphate Turbidity units: 3

LBC Dosage: -

Duration of administration: -

Survival Time: 2 days

Observation Period: 14 days

Diarrhoea days:

0 0	1 +++	2 D	3	4	5	6	7
8	9	10	11	12	13	14	15
16	17	18	19	20	21	22	23
24	25	26	27	28	29	30	31

Body weight changes: Kg.

0 41	7	14	21	28

Cause of death: Pneumonia

Post-mortem findings: Abomasum-normal. Intestine-slight congestion. Lung-congestion. Excessive fluid in joints.

Comments: Signs of meningitis before death.

Calf No. 60 Assigned group: Control Raw Milk

Admission Date: 19.4.1979

Body Wt. (Kg) on admission: 38

Zinc Sulphate Turbidity units: 2

LBC Dosage: -

Duration of administration: -

Survival Time: 7 days

Observation Period: 14 days

Diarrhoea days:

0 0	1 +	2 0	3 +++	4 +++	5 +++	6 +++	7 D
8	9	10	11	12	13	14	15
16	17	18	19	20	21	22	23
24	25	26	27	28	29	30	31

Body weight changes: Kg.

0 38	7	14	21	28

Cause of death: Diarrhoea and pneumonia

Post-mortem findings: Abomasum-congestion. Intestine-congestion and full of air. Lung-congestion. No fluid in joints.

Comments: Umbilical cord inflamed and full of pus.

APPENDIX 8 : SUMMARY OF DATA REGARDING SOURCE, ZST VALUES, WEIGHT CHANGES, GROUP ALLOCATION AND TREATMENT OF 20 INDIVIDUAL CALVES STUDIED IN EXPERIMENT 4

EXPERIMENT 4

TREATMENT			150mgm LBC twice daily in feed; 7 days 75mgm LBC twice daily in feed; 7 days					
Calf No.	Market	ZST	Bwt. (Kg) 8-5	Bwt. (Kg) 16-5	Bwt. (Kg) 23-5	Bwt. (Kg) 30-5	Bwt. (Kg) 6-6	Wt. Gain
High								
61	Lanark	39	38	38	38	41	45	+7
62	Lanark	29	32	33	33	37	41	+9
63	Paisley	23	40	42	39	45	50	+10
Mean		30.3	36.7	37.7	36.7	41.0	45.3	
Intermediate								
64	Lanark	18	33	33	32	36	43	+10
65	Lanark	15	34	33	32	37	40	+6
66	Paisley	13	35	-	-	-	-	-
67	Lanark	12	36	32	-	-	-	-
Mean		14.5	34.5	32.7	32.0	36.5	41.5	
Low								
68	Paisley	9	30	27	-	-	-	-
69	Paisley	7	37	36	36	41	46	+9
70	Paisley	1	32	-	-	-	-	-
Mean		5.7	33.0	31.5	36.0	41.0	46.0	
MEAN		16.6	34.7	34.3	35.0	39.5	44.2	8.5

EXPERIMENT 4

CONTROL

Calf No.	Market	ZST	Bwt.(Kg) 8-5	Bwt.(Kg) 16-5	Bwt.(Kg) 23-5	Bwt.(Kg) 30-5	Bwt.(Kg) 6-6	Wt. Gain
High								
71	Paisley	41	35	36	35	37	40	+5
72	Lanark	28	35	31	27	-	-	-
73	Paisley	23	35	34	32	-	-	-
Mean		30.7	35.0	33.7	31.3	37.0	40.0	
Intermediate								
74	Lanark	23	47	45	42	44	44	-3
75	Lanark	18	38	-	-	-	-	-
76	Lanark	13	31	32	30	30	-	-
77	Lanark	12	36	34	-	-	-	-
Mean		16.5	38.0	37.0	36.0	37.0	44.0	
Low								
78	Lanark	4	30	-	-	-	-	-
79	Lanark	2	29	28	27	28	-	-
80	Lanark	2	32	30	31	30	-	-
Mean		2.7	30.3	29.0	29.0	29.0		
MEAN		16.6	34.8	33.8	32.0	33.8	42.0	

APPENDIX 9 (TABLES 61-80) : INDIVIDUAL DATA SHEETS FOR 20 CALVES USED IN EXPERIMENT 4, INCLUDING INFORMATION REGARDING PRESENCE (AND DEGREE) OR ABSENCE OF DIARRHOEA, SURVIVAL TIME AND GROSS POST MORTEM FINDINGS WHERE APPLICABLE.

Calf No. 61 Assigned group: Treatment Powder Milk

Admission Date: 9.5.1979

Body Wt. (Kg) on admission: 38

Zinc Sulphate Turbidity units: 39

LBC Dosage: 150mg.-feed-twice daily-7 days, then 75mg.-feed-twice daily-7 days.

Duration of administration:

Survival Time: 28 days

Observation Period: 28 days

Diarrhoea days:

0 0	1 0	2 0	3 +	4 +++	5 ++	6 +++	7 0
8 0	9 +	10 0	11 0	12 0	13 0	14 0	15 0
16 0	17 0	18 0	19 0	20 0	21 0	22 +	23 0
24 0	25 0	26 0	27 0	28 0	29	30	31

Body weight changes: Kg.

0 38	7 38	14 38	21 41	28 45

Cause of death: -

Post-mortem findings: -

Comments:

Calf No. 62 Assigned group: Treatment Powder Milk

Admission Date: 9.5.1979

Body Wt. (Kg) on admission: 32

Zinc Sulphate Turbidity units: 29

LBC Dosage: 150mg.-feed-twice daily-7 days, then 75mg.-feed-twice daily-7 days.

Duration of administration:

Survival Time: 28 days

Observation Period: 28 days

Diarrhoea days:

0 0	1 0	2 0	3 0	4 0	5 0	6 ++	7 0
8 0	9 0	10 +++	11 0	12 ++	13 0	14 0	15 0
16 +++	17 0	18 0	19 0	20 0	21 0	22 +	23 0
24 0	25 0	26 0	27 0	28 0	29	30	31

Body weight changes: Kg.

0 32	7 33	14 33	21 37	28 41

Cause of death: -

Post-mortem findings: -

Comments:

Calf No. 63 Assigned group: Treatment Powder Milk

Admission Date: 9.5.1979

Body Wt. (Kg) on admission: 40

Zinc Sulphate Turbidity units: 23

LBC Dosage: 150mg.-feed-twice daily-7 days, then 75mg.-feed-twice daily-7 days.

Duration of administration:

Survival Time: 28 days

Observation Period: 28 days

Diarrhoea days:

0 0	1 0	2 0	3 0	4 0	5 +++	6 +++	7 0
8 0	9 0	10 +++	11 +++	12 0	13 0	14 0	15 0
16 0	17 0	18 0	19 0	20 0	21 0	22 0	23 0
24 0	25 0	26 0	27 0	28 0	29	30	31

Body weight changes: Kg.

0 40	7 42	14 39	21 45	28 50

Cause of death: -

Post-mortem findings: -

Comments:

Calf No. 64 Assigned group: Treatment Powder Milk

Admission Date: 9.5.1979

Body Wt. (Kg) on admission: 33

Zinc Sulphate Turbidity units: 18

LBC Dosage: 150mg.-feed-twice daily-7 days, then 75mg.-feed-twice daily-7 days.

Duration of administration:

Survival Time: 28 days

Observation Period: 28 days

Diarrhoea days:

0 0	1 0	2 0	3 0	4 0	5 ++	6 0	7 +++
8 +++	9 0	10 0	11 0	12 0	13 0	14 0	15 0
16 0	17 0	18 0	19 0	20 0	21 0	22 0	23 0
24 0	25 0	26 0	27 0	28 0	29	30	31

Body weight changes: Kg.

0 33	7 33	14 32	21 36	28 43

Cause of death: -

Post-mortem findings: -

Comments:

Calf No. 65 Assigned group: Treatment Powder Milk

Admission Date: 9.5.1979

Body Wt. (Kg) on admission: 34

Zinc Sulphate Turbidity units: 15

LBC Dosage: 150mg.-feed-twice daily-7 days, then 75mg.-feed-twice daily-7 days.

Duration of administration:

Survival Time: 28 days

Observation Period: 28 days

Diarrhoea days:

0 0	1 0	2 0	3 +++	4 +++	5 0	6 +++	7 +++
8 +++	9 +++	10 0	11 +++	12 +++	13 +++	14 ++	15 +++
16 +++	17 +	18 0	19 0	20 0	21 0	22 0	23 0
24 0	25 0	26 0	27 0	28 0	29	30	31

Body weight changes: Kg.

0 34	7 33	14 32	21 37	28 40

Cause of death: -

Post-mortem findings: -

Comments:

Calf No. 66 Assigned group: Treatment Powder Milk

Admission Date: 9.5.1979

Body Wt. (Kg) on admission: 35

Zinc Sulphate Turbidity units: 13

LBC Dosage: 150mg.-feed-twice daily-7 days, then 75mg.-feed-twice daily-7 days.

Duration of administration:

Survival Time: 4 days

Observation Period: 28 days

Diarrhoea days:

0 0	1 0	2 ++	3 +++	4 D	5	6	7
8	9	10	11	12	13	14	15
16	17	18	19	20	21	22	23
24	25	26	27	28	29	30	31

Body weight changes: Kg.

0 35	7	14	21	28

Cause of death: Bloat and pneumonia

Post-mortem findings: Abomasum-congestion and full of air. Intestine-normal and full of air. Lung-congestion. Excess fluid in joints.

Comments:

Recumbent before death.

Calf No. 67 Assigned group: Treatment Powder Milk

Admission Date: 9.5.1979

Body Wt. (Kg) on admission: 36

Zinc Sulphate Turbidity units: 12

LBC Dosage: 150mg.-feed-twice daily-7 days, then 75mg.-feed-twice daily-7 days.

Duration of administration:

Survival Time: 15 days

Observation Period: 28 days

Diarrhoea days:

0 0	1 0	2 ++	3 +	4 +++	5 +++	6 +++	7 +++
8 ++	9 +++	10 +++	11 +++	12 +++	13 +++	14 0	15 D
16	17	18	19	20	21	22	23
24	25	26	27	28	29	30	31

Body weight changes: Kg.

0 36	7 32	14	21	28

Cause of death: Diarrhoea and pneumonia

Post-mortem findings: Abomasum-congestion and full of air. Intestine-congestion and full of air. Lung-severe congestion.
Comments: No fluid in joints. Rumen-congestion.

Calf No. 68 Assigned group: Treatment Powder Milk

Admission Date: 9.5.1979

Body Wt. (Kg) on admission: 30

Zinc Sulphate Turbidity units: 9

LBC Dosage: 150mg.-feed-twice daily-7 days, then 75mg.-feed-twice daily-7 days.

Duration of administration:

Survival Time: 10 days

Observation Period: 28 days

Diarrhoea days:

0 0	1 0	2 0	3 +++	4 +++	5 +++	6 +++	7 +++
8 ++	9 ++	10 D	11	12	13	14	15
16	17	18	19	20	21	22	23
24	25	26	27	28	29	30	31

Body weight changes: Kg.

0 30	7 27	14	21	28

Cause of death: Diarrhoea and pneumonia

Post-mortem findings: Abomasum-erosion. Intestine-congestion and full of air. Lung-congestion. No fluid in joints. Rumen contained roughage.
Comments:

Calf No. 69 Assigned group: Treatment Powder Milk

Admission Date: 9.5.1979

Body Wt. (Kg) on admission: 37

Zinc Sulphate Turbidity units: 7

LBC Dosage: 150mg.-feed-twice daily-7 days, then 75mg.-feed-twice daily-7 days.

Duration of administration:

Survival Time: 28 days

Observation Period: 28 days

Diarrhoea days:

0 0	1 0	2 +	3 +++	4 +	5 ++	6 0	7 ++
8 +++	9 ++	10 0	11 ++	12 ++	13 ++	14 0	15 0
16 +++	17 0	18 0	19 0	20 0	21 0	22 0	23 0
24 0	25 0	26 0	27 0	28 0	29	30	31

Body weight changes: Kg.

0 37	7 36	14 36	21 41	28 46

Cause of death: -

Post-mortem findings: -

Comments:

Calf No. 70 Assigned group: Treatment Powder Milk

Admission Date: 9.5.1979

Body Wt. (Kg) on admission: 32

Zinc Sulphate Turbidity units: 1

LBC Dosage: 150mg.-feed-twice daily-7 days, then 75mg.-feed-twice daily-7 days.

Duration of administration:

Survival Time: 6 days

Observation Period: 28 days

Diarrhoea days:

0 0	1 0	2 0	3 0	4 +++	5 ++	6 D	7
8	9	10	11	12	13	14	15
16	17	18	19	20	21	22	23
24	25	26	27	28	29	30	31

Body weight changes: Kg.

0 32	7	14	21	28

Cause of death: Diarrhoea

Post-mortem findings: Abomasum-congestion, Intestine-congested and full of air. Lung-normal. Excessive fluid in joints.

Comments:

Calf No. 71 Assigned group: Control Powder Milk

Admission Date: 9.5.1979

Body Wt. (Kg) on admission: 35

Zinc Sulphate Turbidity units: 41

LBC Dosage: -

Duration of administration: -

Survival Time: 28 days

Observation Period: 28 days

Diarrhoea days:

0 0	1 0	2 0	3 0	4 +++	5 0	6 +++	7 +++
8 +++	9 0	10 +++	11 +++	12 ++	13 0	14 0	15 0
16 0	17 0	18 0	19 0	20 0	21 0	22 0	23 0
24 0	25 0	26 0	27 0	28 0	29	30	31

Body weight changes: Kg.

0	7	14	21	28
35	36	35	37	40

Cause of death: -

Post-mortem findings: -

Comments:

Calf No. 72 Assigned group: Control Powder Milk

Admission Date: 9.5.1979

Body Wt. (Kg) on admission: 35

Zinc Sulphate Turbidity units: 28

LBC Dosage: -

Duration of administration: -

Survival Time: 18 days

Observation Period: 28 days

Diarrhoea days:

0 0	1 0	2 0	3 +++	4 +++	5 +++	6 +++	7 +++
8 +++	9 +++	10 +++	11 ++	12 +++	13 +++	14 0	15 0
16 ++	17 0	18 D	19	20	21	22	23
24	25	26	27	28	29	30	31

Body weight changes: Kg.

0	7	14	21	28
35	31	27		

Cause of death: Diarrhoea and pneumonia

Post-mortem findings: Abomasum-congestion and full of air. Intestine-congestion and full of air. Lung-severe congest

Comments: No fluid in joints. Loss of appetite.

Calf No. 73 Assigned group: Control Powder Milk

Admission Date: 9.5.1979

Body Wt. (Kg) on admission: 35

Zinc Sulphate Turbidity units: 23

LBC Dosage: -

Duration of administration: -

Survival Time: 20 days

Observation Period: 28 days

Diarrhoea days:

0 0	1 0	2 0	3 +++	4 +++	5 +++	6 +	7 +++
8 +++	9 +++	10 +++	11 +++	12 +++	13 ++	14 +++	15 ++
16 +++	17 +	18 +++	19 +++	20 D	21	22	23
24	25	26	27	28	29	30	31

Body weight changes: Kg.

0 35	7 34	14 32	21	28

Cause of death: Diarrhoea and pneumonia

Post-mortem findings: Abomasum and Intestine-congestion and full of air. Lung-severe congestion. No fluid in Joints.

Comments:

Calf No. 74 Assigned group: Control Powder Milk

Admission Date: 9.5.1979

Body Wt. (Kg) on admission: 47

Zinc Sulphate Turbidity units: 23

LBC Dosage: -

Duration of administration: -

Survival Time: 28 days

Observation Period: 28 days

Diarrhoea days:

0 0	1 0	2 0	3 0	4 0	5 0	6 0	7 0
8 0	9 0	10 0	11 0	12 +++	13 +++	14 +++	15 +++
16 0	17 +	18 0	19 0	20 0	21 0	22 0	23 0
24 0	25 +	26 0	27 0	28 0	29	30	31

Body weight changes: Kg.

0 47	7 45	14 42	21 44	28 44

Cause of death: -

Post-mortem findings: -

Comments:

Calf No. 75 Assigned group: Control Powder Milk

Admission Date: 9.5.1979

Body Wt. (Kg) on admission: 38

Zinc Sulphate Turbidity units: 18

LBC Dosage: -

Duration of administration: -

Survival Time: 8 days

Observation Period: 28 days

Diarrhoea days:

0 0	1 0	2 0	3 0	4 +++	5 +++	6 +++	7 +++
8 D	9	10	11	12	13	14	15
16	17	18	19	20	21	22	23
24	25	26	27	28	29	30	31

Body weight changes: Kg.

0 38	7	14	21	28

Cause of death: Diarrhoea

Post-mortem findings: Abomasum and Intestine-congestion and full of air. Lung-normal. No fluid in joints.

Comments:

Calf No. 76 Assigned group: Control Powder Milk

Admission Date: 9.5.1979

Body Wt. (Kg) on admission: 31

Zinc Sulphate Turbidity units: 13

LBC Dosage: -

Duration of administration: -

Survival Time: 25 days

Observation Period: 28 days

Diarrhoea days:

0 0	1 0	2 0	3 0	4 0	5 0	6 0	7 ++
8 ++	9 0	10 0	11 0	12 +++	13 +++	14 0	15 0
16 +++	17 0	18 0	19 0	20 0	21 0	22 0	23 0
24 0	25 D	26	27	28	29	30	31

Body weight changes: Kg.

0 31	7 32	14 30	21 30	28

Cause of death: Diarrhoea and pneumonia

Post-mortem findings: Abomasum and Intestine-congestion and full of air. Lung-severe congestion. No fluid in join

Comments: Lacrimation.

Calf No. 77 Assigned group: Control Powder Milk

Admission Date: 9.5.1979

Body Wt. (Kg) on admission: 36

Zinc Sulphate Turbidity units: 12

LBC Dosage: -

Duration of administration: -

Survival Time: 9 days

Observation Period: 28 days

Diarrhoea days:

0 0	1 +	2 0	3 +	4 0	5 0	6 0	7 +++
8 +++	9 D	10	11	12	13	14	15
16	17	18	19	20	21	22	23
24	25	26	27	28	29	30	31

Body weight changes: Kg.

0 36	7 34	14	21	28

Cause of death: Pneumonia

Post-mortem findings: Abomasum-normal. Intestine-congestion. Lung-severe congestion. No fluid in joints.

Comments: Coughing.

Calf No. 78 Assigned group: Control Powder Milk

Admission Date: 9.5.1979

Body Wt. (Kg) on admission: 30

Zinc Sulphate Turbidity units: 4

LBC Dosage: -

Duration of administration: -

Survival Time: 4 days

Observation Period: 28 days

Diarrhoea days:

0 0	1 +++	2 +++	3 +++	4 D	5	6	7
8	9	10	11	12	13	14	15
16	17	18	19	20	21	22	23
24	25	26	27	28	29	30	31

Body weight changes: Kg.

0 30	7	14	21	28

Cause of death: Diarrhoea and pneumonia

Post-mortem findings: Abomasum-congestion. Intestine-congestion and full of air. Lung-congestion. Excessive fluid in joints.

Comments: Loss of appetite.

Calf No. 79 Assigned group: Control Powder Milk

Admission Date: 9.5.1979

Body Wt. (Kg) on admission: 29

Zinc Sulphate Turbidity units: 2

LBC Dosage: -

Duration of administration: -

Survival Time: 28 days

Observation Period: 28 days

Diarrhoea days:

0 0	1 0	2 +	3 0	4 +++	5 +++	6 +++	7 +++
8 +++	9 +++	10 +++	11 +++	12 +++	13 +++	14 +++	15 ++
16 +++	17 +++	18 +++	19 0	20 0	21 0	22 0	23 +
24 0	25 0	26 +	27 ++	28 D	29	30	31

Body weight changes: Kg.

0 29	7 28	14 27	21 28	28

Cause of death: Pneumonia and diarrhoea

Post-mortem findings: Abomasum and Intestine-congestion and full of air. Lung-severe congestion. No fluid in joints.

Comments:

Calf No. 80 Assigned group: Control Powder Milk

Admission Date: 9.5.1979

Body Wt. (Kg) on admission: 32

Zinc Sulphate Turbidity units: 2

LBC Dosage: -

Duration of administration: -

Survival Time: 28 days

Observation Period: 28 days

Diarrhoea days:

0 0	1 +++	2 +	3 +++	4 +++	5 +++	6 +++	7 +++
8 ++	9 +++	10 ++	11 ++	12 0	13 0	14 0	15 +++
16 0	17 0	18 0	19 ++	20 +	21 0	22 0	23 0
24 0	25 0	26 0	27 0	28 D	29	30	31

Body weight changes: Kg.

0 32	7 30	14 31	21 30	28

Cause of death: Pneumonia and diarrhoea

Post-mortem findings: Abomasum and Intestine-congestion and full of air. Lung-severe congestion. No fluid in joint

Comments:

APPENDIX 10 : SUMMARY OF DATA REGARDING SOURCE, ZST VALUES, WEIGHT CHANGES, GROUP ALLOCATION AND TREATMENT OF 16 INDIVIDUAL CALVES STUDIED IN EXPERIMENT 5

EXPERIMENT 5

RECONSTITUTED (BOCM) MILK POWDER 150mgm LBC twice daily in feed; 7 days 75mgm LBC twice daily in feed; 7 days								RAW MILK 150mgm LBC twice daily in feed; 7 days 75mgm LBC twice daily in feed; 7 days							
Calf No.	Market	ZST	Bwt. (Kg) 25-7	Bwt. (Kg) 1-8	Bwt. (Kg) 8-8	Bwt. (Kg) 15-8	Wt. Gain	Calf No.	Market	ZST	Bwt. (Kg) 25-7	Bwt. (Kg) 1-8	Bwt. (Kg) 8-8	Bwt. (Kg) 15-8	Wt. Gain
High								High							
81	Ayr	40	36	37	35	42	+6	89	Ayr	38	34	36	37	42	+8
82	Ayr	36	34	32	29	30	-4	90	Lanark	38	33	35	39	44	+11
83	Lanark	33	32	30	30	33	+1	91	Lanark	35	35	36	36	45	+10
Mean		36.3	34.0	33.0	31.3	35.0		Mean		37.0	34.0	35.7	37.3	43.7	
Intermediate								Intermediate							
84	Lanark	28	37	38	40	45	+8	92	Ayr	30	36	-	-	-	-
85	Ayr	28	30	27	23	-	-	93	Lanark	28	40	37	39	42	+2
Mean		28	33.5	32.5	31.5	45.0		Mean		29.0	38.0	37.0	39.0	42.0	
Low								Low							
86	Paisley	27	41	40	36	42	+1	94	Ayr	26	40	37	-	-	-
87	Ayr	26	27	29	26	-	-	95	Ayr	23	23	23	26	29	+6
88	Ayr	21	35	-	-	-	-	96	Lanark	21	33	36	43	47	+14
Mean		24.7	34.3	34.5	31.0	42.0		Mean		23.3	32.0	32.0	34.5	38.0	
MEAN		29.9	34.0	33.3	31.3	38.4	2.4	MEAN		29.8	34.3	34.3	36.7	41.5	8.5

APPENDIX 11 (TABLES 81-96) : INDIVIDUAL DATA SHEETS FOR 16 CALVES USED IN EXPERIMENT 5, INCLUDING INFORMATION REGARDING PRESENCE (AND DEGREE) OR ABSENCE OF DIARRHOEA, SURVIVAL TIME AND GROSS POST MORTEM FINDINGS WHERE APPLICABLE

Calf No. 81 Assigned group: Treatment Powder Milk

Admission Date: 25.7.1979

Body Wt. (Kg) on admission: 36

Zinc Sulphate Turbidity units: 40

LBC Dosage: 150mg.-feed-twice daily-7 days, then 75mg.-feed-twice daily-7 days.

Duration of administration:

Survival Time: 21 days

Observation Period: 21 days

Diarrhoea days:

0 0	1 0	2 0	3 0	4 0	5 0	6 0	7 +++
8 +++	9 +++	10 +++	11 ++	12 +++	13 +++	14 0	15 0
16 0	17 0	18 0	19 0	20 0	21 0	22	23
24	25	26	27	28	29	30	31

Body weight changes: Kg.

0 36	7 37	14 35	21 42	28

Cause of death: -

Post-mortem findings: -

Comments:

Calf No. 82 Assigned group: Treatment Powder Milk

Admission Date: 25.7.1979

Body Wt. (Kg) on admission: 34

Zinc Sulphate Turbidity units: 36

LBC Dosage: 150mg.-feed-twice daily-7 days, then 75mg.-feed-twice daily-7 days.

Duration of administration:

Survival Time: 21 days

Observation Period: 21 days

Diarrhoea days:

0 0	1 0	2 0	3 0	4 0	5 ++	6 +++	7 0
8 0	9 ++	10 0	11 0	12 0	13 0	14 0	15 0
16 0	17 0	18 0	19 +	20 0	21 +++	22	23
24	25	26	27	28	29	30	31

Body weight changes: Kg.

0 34	7 32	14 29	21 30	28

Cause of death: -

Post-mortem findings: -

Comments:

Calf No. 83 Assigned group: Treatment Powder Milk

Admission Date: 25.7.1979

Body Wt. (Kg) on admission: 32

Zinc Sulphate Turbidity units: 33

LBC Dosage: 150mg.-feed-twice daily-7 days, then 75mg.-feed-twice daily-7 days.

Duration of administration:

Survival Time: 21 days

Observation Period: 21 days

Diarrhoea days:

0 0	1 ++	2 0	3 ++	4 +++	5 0	6 +++	7 +++
8 +++	9 +++	10 +++	11 +++	12 +++	13 +++	14 +++	15 +++
16 +++	17 +++	18 +	19 +++	20 +++	21 +++	22	23
24	25	26	27	28	29	30	31

Body weight changes: Kg.

0	7	14	21	28
32	30	30	33	

Cause of death: -

Post-mortem findings: -

Comments:

Calf No. 84 Assigned group: Treatment Powder Milk

Admission Date: 25.7.1979

Body Wt. (Kg) on admission: 37

Zinc Sulphate Turbidity units: 28

LBC Dosage: 150mg.-feed-twice daily-7 days, then 75mg.-feed-twice daily-7 days.

Duration of administration:

Survival Time: 21 days

Observation Period: 21 days

Diarrhoea days:

0 0	1 0	2 0	3 0	4 ++	5 +++	6 0	7 0
8 +++	9 ++	10 0	11 0	12 0	13 0	14 0	15 0
16 0	17 0	18 0	19 0	20 0	21 0	22	23
24	25	26	27	28	29	30	31

Body weight changes: Kg.

0	7	14	21	28
37	38	40	45	

Cause of death: -

Post-mortem findings: -

Comments:

Calf No. 85 Assigned group: Treatment Powder Milk

Admission Date: 25.7.1979

Body Wt. (Kg) on admission: 30

Zinc Sulphate Turbidity units: 28

LBC Dosage: 150mg.-feed-twice daily-7 days, then 75mg.-feed-twice daily-7 days.

Duration of administration:

Survival Time: 20 days

Observation Period: 21 days

Diarrhoea days:

0 0	1 0	2 ++	3 0	4 +++	5 +++	6 +++	7 +++
8 +++	9 +++	10 +++	11 +++	12 0	13 0	14 0	15 0
16 0	17 +	18 ++	19 0	20 D	21	22	23
24	25	26	27	28	29	30	31

Body weight changes: Kg.

0 30	7 27	14 23	21	28

Cause of death: Pneumonia and diarrhoea

Post-mortem findings: Abomasum-normal. Intestine-congestion and full of air. Lung-severe congestion. Excessive fluid in joints.

Comments: Dehydration.

Calf No. 86 Assigned group: Treatment Powder Milk

Admission Date: 25.7.1979

Body Wt. (Kg) on admission: 41

Zinc Sulphate Turbidity units: 27

LBC Dosage: 150mg.-feed-twice daily-7 days, then 75mg.-feed-twice daily-7 days.

Duration of administration:

Survival Time: 21 days

Observation Period: 21 days

Diarrhoea days:

0 0	1 0	2 0	3 0	4 0	5 0	6 +++	7 +++
8 +++	9 +++	10 ++	11 ++	12 +++	13 +++	14 0	15 ++
16 0	17 +	18 ++	19 +++	20 0	21 0	22	23
24	25	26	27	28	29	30	31

Body weight changes: Kg.

0 41	7 40	14 36	21 42	28

Cause of death: -

Post-mortem findings: -

Comments:

Calf No. 87 Assigned group: Treatment Powder Milk

Admission Date: 25.7.1979

Body Wt. (Kg) on admission: 27

Zinc Sulphate Turbidity units: 26

LBC Dosage: 150mg.-feed-twice daily-7 days, then 75mg.-feed-twice daily-7 days.

Duration of administration:

Survival Time: 21 days

Observation Period: 21 days

Diarrhoea days:

0 0	1 0	2 0	3 0	4 ++	5 +++	6 ++	7 ++
8 +++	9 0	10 +++	11 +	12 ++	13 +++	14 ++	15 ++
16 0	17 ++	18 +++	19 0	20 +++	21 D	22	23
24	25	26	27	28	29	30	31

Body weight changes: Kg.

0	7	14	21	28
27	29	26		

Cause of death: Pneumonia and diarrhoea

Post-mortem findings: Abomasum-normal. Intestine-congestion and full of air. Lung-severe congestion. No fluid in joints.

Comments: Dehydration.

Calf No. 88 Assigned group: Treatment Powder Milk

Admission Date: 25.7.1979

Body Wt. (Kg) on admission: 35

Zinc Sulphate Turbidity units: 21

LBC Dosage: 150mg.-feed-twice daily-7 days, then 75mg.-feed-twice daily-7 days.

Duration of administration:

Survival Time: 7 days

Observation Period: 21 days

Diarrhoea days:

0 0	1 0	2 0	3 +++	4 +++	5 +++	6 +++	7 D
8	9	10	11	12	13	14	15
16	17	18	19	20	21	22	23
24	25	26	27	28	29	30	31

Body weight changes: Kg.

0	7	14	21	28
35				

Cause of death: Diarrhoea

Post-mortem findings: Abomasum and Intestine-congestion and full of air. Lung-normal. Excessive fluid in joints.

Comments: Dehydration.

Calf No. 89 Assigned group: Treatment Raw Milk

Admission Date: 25.7.1979

Body Wt. (Kg) on admission: 34

Zinc Sulphate Turbidity units: 38

LBC Dosage: 150mg.-feed-twice daily-7 days, then 75mg.-feed-twice daily-7 days.

Duration of administration:

Survival Time: 21 days

Observation Period: 21 days

Diarrhoea days:

0 0	1 0	2 0	3 0	4 0	5 0	6 +	7 0
8 0	9 0	10 0	11 0	12 0	13 0	14 0	15 0
16 0	17 0	18 0	19 0	20 0	21 0	22	23
24	25	26	27	28	29	30	31

Body weight changes: Kg.

0 34	7 36	14 37	21 42	28

Cause of death: -

Post-mortem findings: -

Comments:

Calf No. 90 Assigned group: Treatment Raw Milk

Admission Date: 25.7.1979

Body Wt. (Kg) on admission: 33

Zinc Sulphate Turbidity units: 38

LBC Dosage: 150mg.-feed-twice daily-7 days, then 75mg.-feed-twice daily-7 days.

Duration of administration:

Survival Time: 21 days

Observation Period: 21 days

Diarrhoea days:

0 0	1 0	2 +++	3 0	4 0	5 0	6 0	7 0
8 0	9 0	10 0	11 0	12 0	13 0	14 0	15 0
16 0	17 0	18 0	19 0	20 0	21 0	22	23
24	25	26	27	28	29	30	31

Body weight changes: Kg.

0 33	7 35	14 39	21 44	28

Cause of death: -

Post-mortem findings: -

Comments:

Calf No. 91 Assigned group: Treatment Raw Milk

Admission Date: 25.7.1979

Body Wt. (Kg) on admission: 35

Zinc Sulphate Turbidity units: 35

LBC Dosage: 150mg.-feed-twice daily-7 days, then 75mg.-feed-twice daily-7 days.

Duration of administration:

Survival Time: 21 days

Observation Period: 21 days

Diarrhoea days:

0 0	1 0	2 +++	3 +++	4 +++	5 ++	6 +++	7 0
8 +++	9 +	10 0	11 ++	12 0	13 +++	14 0	15 0
16 0	17 0	18 0	19 0	20 0	21 0	22	23
24	25	26	27	28	29	30	31

Body weight changes: Kg.

0 35	7 36	14 36	21 45	28

Cause of death: -

Post-mortem findings: -

Comments:

Calf No. 92 Assigned group: Treatment Raw Milk

Admission Date: 25.7.1979

Body Wt. (Kg) on admission: 36

Zinc Sulphate Turbidity units: 30

LBC Dosage: 150mg.-feed-twice daily-7 days, then 75mg.-feed-twice daily-7 days.

Duration of administration:

Survival Time: 8 days

Observation Period: 21 days

Diarrhoea days:

0 0	1 0	2 0	3 0	4 0	5 +++	6 +++	7 +++
8 D	9	10	11	12	13	14	15
16	17	18	19	20	21	22	23
24	25	26	27	28	29	30	31

Body weight changes: Kg.

0 36	7	14	21	28

Cause of death: Diarrhoea

Post-mortem findings: Abomasum and Intestine-congestion and full of air. Lung-normal. No fluid in joints.

Comments: Dehydration.

Calf No. 93 Assigned group: Treatment Raw Milk

Admission Date: 25.7.1979

Body Wt. (Kg) on admission: 40

Zinc Sulphate Turbidity units: 28

LBC Dosage: 150mg.-feed-twice daily-7 days, then 75mg.-feed-twice daily-7 days.

Duration of administration:

Survival Time: 21 days

Observation Period: 21 days

Diarrhoea days:

0 0	1 0	2 0	3 0	4 0	5 +++	6 +++	7 0
8 0	9 0	10 +++	11 0	12 +++	13 0	14 0	15 0
16 0	17 0	18 0	19 0	20 +++	21 +	22	23
24	25	26	27	28	29	30	31

Body weight changes: Kg.

0 40	7 37	14 39	21 42	28

Cause of death: -

Post-mortem findings: -

Comments:

Calf No. 94 Assigned group: Treatment Raw Milk

Admission Date: 25.7.1979

Body Wt. (Kg) on admission: 40

Zinc Sulphate Turbidity units: 26

LBC Dosage: 150mg.-feed-twice daily-7 days, then 75mg.-feed-twice daily-7 days.

Duration of administration:

Survival Time: 9 days

Observation Period: 21 days

Diarrhoea days:

0 0	1 0	2 0	3 +++	4 +++	5 +++	6 +++	7 +++
8 +++	9 D	10	11	12	13	14	15
16	17	18	19	20	21	22	23
24	25	26	27	28	29	30	31

Body weight changes: Kg.

0 40	7 37	14	21	28

Cause of death: Pneumonia and diarrhoea

Post-mortem findings: Abomasum-normal. Intestine-congestion and full of air. Lung-severe congestion. No fluid in joints.

Comments:

Calf No. 95 Assigned group: Treatment Raw Milk

Admission Date: 25.7.1979

Body Wt. (Kg) on admission: 23

Zinc Sulphate Turbidity units: 23

LBC Dosage: 150mg.-feed-twice daily-7 days, then 75mg.-feed-twice daily-7 days.

Duration of administration:

Survival Time: 21 days

Observation Period: 21 days

Diarrhoea days:

0 0	1 0	2 +++	3 0	4 +++	5 +++	6 0	7 0
8 0	9 +	10 0	11 0	12 0	13 0	14 0	15 0
16 0	17 0	18 0	19 0	20 0	21 0	22 0	23
24	25	26	27	28	29	30	31

Body weight changes: Kg.

0 23	7 23	14 26	21 29	28

Cause of death: -

Post-mortem findings: -

Comments:

Calf No. 96 Assigned group: Treatment Raw Milk

Admission Date: 25.7.1979

Body Wt. (Kg) on admission: 33

Zinc Sulphate Turbidity units: 21

LBC Dosage: 150mg.-feed-twice daily-7 days, then 75mg.-feed-twice daily-7 days.

Duration of administration:

Survival Time: 21 days

Observation Period: 21 days

Diarrhoea days:

0 0	1 0	2 0	3 +++	4 +++	5 0	6 0	7 ++
8 0	9 0	10 0	11 0	12 0	13 0	14 0	15 0
16 +++	17 0	18 0	19 0	20 0	21 0	22	23
24	25	26	27	28	29	30	31

Body weight changes: Kg.

0 33	7 36	14 43	21 47	28

Cause of death: -

Post-mortem findings: -

Comments: